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journal of dental medicine

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(Quoted from the Constitution)

"To promote the study and dissemination of knowledge of the cause, prevention and control of diseases of the teeth, their supporting structures and adnexa and related subjects; and to foster and promote a better scientific understanding between the fields of dentistry and medicine."

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1. Manuscripts should be typed, double spaced, on one side of 8½ by 11 inch bond paper, allowing one inch margins.

2. The title should appear at the top of the first page, followed by the name and degrees of the author or authors. The address of the author should appear at the end of the article.

3. The name of the group before which the paper was presented, the date and place of delivery and the author's university, hospital or government affiliation, if any, should appear at the bottom of the title page.

4. Manuscripts presented for publication should: a—present new facts, describe new techniques or methods of practice or reevaluate established principles; b—contain the results of original research, or c—provide a review of facts concerning a particular subject so that the reader may deduce therefrom legitimate, important conclusions.

5. Data presented in charts and tables should be discussed but not repeated in the text.

6. A concise summary and a precise conclusion add to the value of a scientific article. The summary should state briefly the purpose of the article and the main facts presented or reviewed.

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JOURNAL OF DENTAL MEDICINE

IS DEDICATED TO THE MEMORY OF

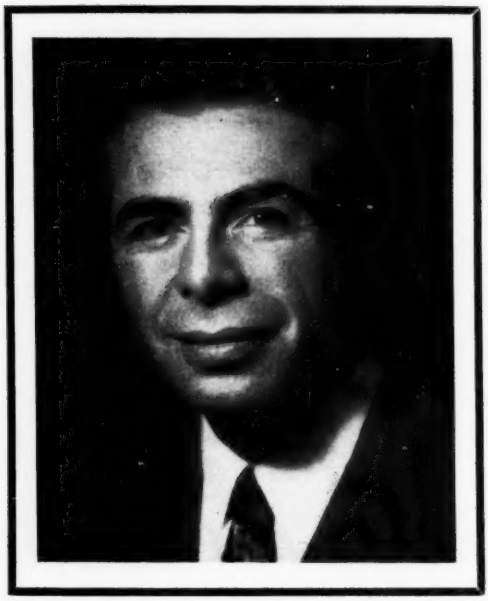
Doctor Samuel Charles Miller

FOUNDER, FIRST PRESIDENT AND INSPIRING LEADER

OF THE

AMERICAN ACADEMY OF DENTAL MEDICINE

DOCTOR SAMUEL CHARLES MILLER



Doctor Samuel Charles Miller, founder and first president of the American Academy of Dental Medicine, passed away after a brief illness on February 8th, 1958. His sudden death deeply shocked his many friends and colleagues all over the world. He was 54 years old.

Until his demise, Dr. Miller was Professor and Chairman of the Department of Periodontia and Oral Medicine at New York University College of Dentistry. Other associations included Attending in Oral Medicine, Polyclinic Hospital and Medical School; Consultant in Oral Medicine, Jewish Chronic Disease Hospital; Consultant in Oral Medicine, Veterans Administration; Consultant and Postgraduate Teacher, United States Navy; Consultant in Oral Medicine, University Hospital; Diplomate, American Board of Periodontology, and Chairman, American Board of Oral Medicine.

In his youth, Dr. Miller attended the Townsend Harris Hall High School and the College of the City of New York. He was graduated from New York University College of Dentistry in 1925. During his Junior and Senior years at the Dental College, Dr. Miller taught physiology and histology as an undergraduate instructor. From 1925 to 1928 he was an Instructor in Periodontia at the Dental College, and was Chief of the Diagnostic Department from 1928 to 1930. During the next two years, Dr. Miller was Assistant Professor of Periodontia. Between 1932 and 1947, he served as Associate Pro-

fessor and Chairman of the Periodontia Department. Since 1947, Dr. Miller had been Professor and Chairman of the Department of Periodontia and Oral Medicine.

With Dr. Sidney Sorrin, he instituted postgraduate courses in Periodontia and Oral Medicine at New York University College of Dentistry and trained over 600 postgraduate students from 43 states and more than 40 foreign countries as teachers, practitioners and specialists in his field.

Dr. Miller was a Fellow of the American College of Dentists, American Academy of Dental Medicine, American Association for the Advancement of Science and an Associate Fellow of the New York Academy of Medicine. Beside being a past president and founder of the American Academy of Dental Medicine, Dr. Miller was past president of both Omicron Kappa Upsilon (Omega Chapter) and the Alumni Association of New York University College of Dentistry. He was also president-elect of the Pan American Odontologic Association.

Dr. Miller was the recipient of many awards during his career. Upon graduation he received the Faneuil D. Weiss award for excellence in scholarship. He was an Honorary Professor of the University of Santo Domingo Dental School and an honorary member of the Bolivian Dental Association, El Primer Congreso Nacional de Odontologos (Colombia, S.A.) the Northwestern Dental Alumni Association and the Alpha Omega Fraternity.

In addition to his membership in national, state and local dental societies, Dr. Miller was an active member of the New York Academy of Science, the American Academy of Periodontology, The Scientific Research Society of America, the International Association of Dental Research and the Harvey Society.

He was an author of four books and a contributor to several more. The first book "Practice of Periodontia" by Dr. Sidney Sorrin and Dr. Miller was published in 1928. Within ten years he had authored two additional texts, "Oral Diagnosis and Treatment," and "Textbook of Periodontia." Both of these texts are now in the third edition. In press at present is the "Spanish-English, Medical-Dental Interpreter" which should be invaluable to physicians and dentists who wish to converse with patients in Spanish.

On the national and international scene, Dr. Miller lectured throughout the United States before dental societies and in many dental schools. He had an excellent command of foreign languages, and when in France, lectured in French; in Italy, in Italian; and in Spain and South America, in Spanish. A world-wide traveler, he spread his knowledge of Periodontia throughout the world and stimulated advanced periodontal studies and research wherever he lectured. Many of the articles that he wrote were translated into several foreign languages. His two textbooks have recently appeared in Spanish editions and are widely read.

Dr. Miller's passing is a great loss to organized dentistry, especially New York University College of Dentistry and the American Academy of Dental Medicine. He was a devoted husband, father and brother. Surviving are his widow, Mrs. Mae Rodgers Miller; two daughters, Mrs. Barbara J. Marsak and Mrs. Stephanie A. Nevins; two sisters, Rose and Dorothy; and two brothers, Harry and David.

The officers and members of the American Academy of Dental Medicine extend their heartfelt and deepest sympathy to the bereaved family.

A WORLD-WIDE SURVEY OF ACUTE NECROTIZING ULCERATIVE GINGIVITIS: A PRELIMINARY REPORT

SAMUEL CHARLES MILLER, D.D.S., F.A.C.D., F.A.D.M.*

and

HENRY I. GREENE, B.A., D.D.S.†

The search for etiologic factors and definitive treatment of acute necrotizing ulcerative gingivitis has been filled with controversy. While the etiology of the disease has been attributed to many different factors — contagion, seasonal, psychosomatic, bacterial, nutritional, etc., the methods of treatment and medication suggested have been fantastically numerous. As yet, a clear-cut concept of the etiology and treatment of the disease is not proven. This tremendous variance of opinion has led to confusion which has obscured the areas of agreement from which rational concepts might be postulated.

It was for this reason that it was decided to initiate a definitive statistical study, the data of which could become the foundation of basic scientific research in the field. Our first attempt‡ was to send detailed questionnaires to various schools around the world. We had hoped to cull from the records of the five preceding years a correlation between the incidence of acute necrotizing ulcerative gingivitis and climatic and seasonal variations. However, we found that because (1) many of the schools had incomplete records due to the war and (2) each school has a different manner of recording data, we were unable to obtain the information necessary to formulate any valid conclusions.

With this failure to gain reliable data from previous records, it was decided to organize a new study in which the data would be collected on a standardized chart. (Fig. I) The chart was devised with the purpose of (1) embracing all the controversial facets involved in etiology and treatment and, (2) collecting data which might uncover statistically-interesting predilections of the disease. The following paragraphs are a discussion of the rationale, based on a study of the literature, for selecting the various components of the chart. It is hoped that data, carefully recorded on these charts, will furnish comprehensive information and sound conclusions.

NAME OF PATIENT

Although most of the data columns on the chart will be filled in on the patient's initial visit, there may be some data that the clinician might wish to add on succeeding visits—e.g. date of dismissal, additional comments, method of treatment, etc. For this reason, we felt it would be advisable to use the patient's name, so that the clinician could use it as a quick means to locate that patient's data line for later entries. Another reason for using the patient's name is to use it as a means of checking back on any recurrences of the disease in a particular patient. Since the study will continue for a period of five years, there is a possibility that the condition may recur in some patients. If a recurrence does occur, the clinician should treat it as a separate case line. (It would be helpful, if the clinician is aware that the patient is returning with a recurrence of the disease within the five year span of the study, if he would mark an asterick next to the patient's name.) By keeping the data organized in this fashion, the study can serve on both a statistical level and as a basis for more minute detailed evaluation.

* Professor and Chairman of the Periodontia and Oral Medicine Department, New York University College of Dentistry.

† Instructor in Periodontia and Oral Medicine, New York University College of Dentistry.

‡ We wish to express our deep appreciation to Norman Alderman, D.D.S. who extended great effort in the first part of this study. Although the data obtained was not published, it helped greatly in creating the format for this project.

SEX

Most statistical and clinical studies of acute necrotizing ulcerative gingivitis have been carried out among male army personnel. (Bouty, (1917); Brooks and Wilson, (1944); Campbell and Dyas, (1917); Carter and Ball, (1953); Cofield et al, (1945); Goadby, (1916); Grupe and Wilder, (1956); Hester et al, (1952); Ludwick, (1944); MacGregor and Long, (1945); Naegeli and Morginson, (1945); Pearce and MacDonald, (1945); Pindborg, (1951); Reckord and Baker, (1920); Schluger, (1949); Schuessler et al, (1945); Strock, (1945); Thexton and Dick, (1945); Wihr, (1946). Fish, (1938) states his belief that the disease occurs principally in men, yet Miller and Greenhut (1944), in a study of 127 clinic patients, had almost an equal distribution among male and female patients. Laughlen et al (1919), in a report on a study on civilian population, found that 10 to 15% of 150 cases studied had acute necrotizing ulcerative gingivitis and of these 15 to 20 cases, 10 were female. Stammers (1944) in a civilian study on 1,017 cases found "both sexes almost equally affected" (503 males and 514 females). Wilkie (1945), however, examining a civilian population of approximately 10,000, studying 200 cases closely, makes no reference to a differential occurence between the sexes. This study, since it is concerned principally with civilian populations, should disclose any statistically signficant difference in the occurrence of the disease on the basis of sex.

RACE

It has been demonstrated that some disease entities occur more readily in one racial group in contrast to another. The reasons are various —greater susceptibility because of environmental conditions, nutritional habits, climatic factors, etc. There has been very little evaluation of this facet in the literature. Brucker (1956) in a study of 2542 children for incidence of acute necrotizing ulcerative gingivitis found there was a higher number of positive smears in the Negro children than in the white children. (Significance of this finding depends upon our acceptance of a diagnosis of acute necrotizing ulcerative gingivitis based on a positive smear alone.) It would be of interest to determine, in this study, whether the disease demonstrates any racial propensity.

AGE

During the time in which a nation is at war, the greatest bulk of its draftees falls between the ages of 18-30. Because the majority of studies of acute necrotizing ulcerative gingivitis have been conducted on men in the armed forces under wartime conditions, it is not surprising that the age group that has been quoted as most susceptible to acute necrotizing ulcerative gingivitis is from 18-30. (E.g. Brooks and Wilson (1944) state the ages of the men treated were mostly between 17 and 25; Reckord and Baker (1920) give the average age as 24½.) However, civilian group studies such as Goodrow (1936) states that in an "outbreak" in the San Luiz Valley, the disease appeared in children suffering from deficiency diseases and bad infections. Brucker (1956), although he found a greater prevalence among 12 to 13 year old children, feels that there is no relation between age and the occurrence of the disease. On the other hand, Miller and Greenhut (1944) found the greatest incidence of acute necrotizing ulcerative gingivitis among patients of a university dental clinic from 15 to 30 years of age, the peak occuring between the ages of 19 and 22. Stammers (1944) in a civillian study, had patients from 3 to 68 years old. The age distribution, however, resembled a bell curve, having the greatest percentage (66.96 per cent) of patients in the 19 to 30 year age range. Since this will be the first intensive large scale civilian study, any statistically significant predilection of the disease for a particular age group (if it exists) should be uncovered.

OCCUPATION

There has been no evaluation in the literature of any correlation between occupation and the occurence of acute necrotizing ulcerative gingivitis. True, there has been high incidence of the disease among army personnel, but chiefly under the stress conditions of war. The authors have been unable to find any reports in the litera-

ture of a high incidence of the disease in peacetime personnel. Another group that has shown a high susceptibility are college students (Pardee et al, 1928). These two groups are made up of people subject to stress. No mention has been made in the literature of the occupation of patients studied other than students and soldiers.

It would be of interest to note what percentage of the patients, during this five year study, work in stress-producing occupations and whether any correlation can be deduced from such data.

DEGREE OF SEVERITY

There are two reasons for indicating data on degree of severity. Firstly, it gives a clue to the patient's resistance to the disease and may help in evaluating any recurrence of the disease in that particular patient. It might also give an indication of any possible correlation between the type of etiologic factor and any effect this might have on the severity of the condition. Secondly, it would aid in judging the efficacy of the method of treatment employed in the case. The results of treatment of a mild case should not be judged in the same terms as that for a severe form of the disease. In this way by having one more facet of information, we will be able to more accurately interpret our data.

DATE OF ONSET—DATE OF DISMISSAL

Our reason for desiring data for the date of onset and date of dismissal is to find if there are any variations in the duration of the disease and if so, if these differences can be correlated to different etiologic factors or methods of treatment.

EVALUATION OF ORAL CLEANLINESS

A poor state of oral cleanliness has been a fairly well accepted factor in the etiology of acute necrotizing ulcerative gingivitis. The Research Commission of the ADA (1945) in regarding oral sepsis and irritants as causative factors with concommitant growth in the number of Vincent organisms concurred with Pilot (1928) who found a greater number of Vincent's organisms in dirty mouths. Farrell and McNichols (1937) stated "Its main requirement to appear in epidemic form is poor mouth hygiene and crowded quarters." Schnurman (1947) felt the outbreak he observed was due to faulty dental hygiene, whereas Cripps (1953) believed that acute necrotizing ulcerative gingivitis occurred in poor periodontal mouths and in filthy mouths. Brooks and Wilson (1944), who agreed it is a disease of filth, stated that "oral hygiene, for coping with the problem, tells 90 per cent of the story", and Pardee et al (1928), in reporting an "outbreak" of the disease at a girl's college, felt that good oral hygiene seemed to be the most important factor in decreasing susceptibility. Miller (1950) postulates that "whenever uncleanliness exists together with a laxity in oral sanitation measures, there is certain to be a lowering of tissue resistance and consequently a tendency toward the initiation of necrotizing ulcerative gingivitis."

The data collected on the state of oral cleanliness of the patient on presentation will be used in correlation against degree of severity, duration of disease and the influence of other etiologic factors. It will also be important in evaluating methods of treatment.

NUTRITIONAL STATUS OF PATIENT

Goodrow (1936) in reporting on an "epidemic" of acute necrotizing ulcerative gingivitis in the San Luiz Valley found that 80% of malnourished individuals were affected and concluded that a deficiency diet renders one more susceptible to Vincent's organisms. Box and Ham (1942) postulated a causal relationship between ascorbic acid deficiency and acute necrotizing ulcerative gingivitis. Roth and Stone (1951) and Reckord and Baker (1920) believed malnutrition may be considered a contributary cause; and Cripps (1953) states that acute necrotizing ulcerative gingivitis occurs in individuals lacking a diet high in vitamins.

Although there is a fairly general acceptance of the fact that poor nutriture is a predisposing factor in the initiation of acute necrotizing ulcerative gingivitis, the specifics seem to be in great controversy. Kirkpatrick (1939), in examining 2,000 natives in Australia, found a partial de-

ficiency of Vitamin A and B in conjunction with a high carbohydrate diet was associated with Vincent's infection. Sinclair (1939) agrees that in regard to Vitamin A and B "clinical and subclinical avitaminosis cases may have a direct relationship to Vincent's". On the other hand, Wilkie (1945) felt that Vitamin A and B were not a predisposing factor and treated 42 cases with Vitamin C, getting 50% results. Weisberger (1941), using 200 mg. of nicotinic acid daily, claimed to observe a "cleaning up" of Vincent's after 36 hours, whereas Miller (1950) states "experimentation with nicotinic acid showed no improvement of the clinical oral picture in typical acute necrotizing ulcerative gingivitis." Instead he recommends the daily administration of 500 mg. of ascorbic acid and 5 to 15 mg. of riboflavin. Roth (1945) cites a case in which 1500 mg. of Vitamin C were administered for two days with dramatic results and Schnurman (1947) recommends massive doses of vitamin C too, while Smith (1942) prescribed only 50 mg of Vitamin C three times daily.

Knowledge of the nutritional status of the patient in this study will aid us in evaluating other aspects of the data. If the nutritional state of the patient is poor, and the clinician is aware of specific deficiencies diagnostically, it is hoped that the clinician will make a note of his observations.

NUMBER OF PREVIOUS EXPERIENCES WITH DISEASE

The data obtained on the patient's previous experiences with the disease will give an indication of percentage of recurrences, whether certain etiologic factors predispose a patient to recurrence more than others, and whether certain methods of treatment are more efficacious than others in clearing up the oral condition completely. The cases in which recurrences will take place during the five year period of the study will also aid us in comparing the two or more records of the patient to study any similarities or differences between the various experiences the patient has with the disease.

CONTAGION

Historically, the deleterious effect of acute necrotizing ulcerative gingivitis on the efficiency of soldiers has been recorded from 401 B.C., when Xenophen's army, during an invasion of Asia, was forced to retreat because the men were suffering from sore, foul-smelling mouths. Since the symptoms appeared almost simultaneously among large numbers of soldiers living closely together, the earliest thinking was that acute necrotizing ulcerative gingivitis was a highly contagious disease. With the publication of Plaut's (1894) and Vincent's (1896) detailed description of the spirochete and fusiform bacillus, and the implication of these organisms with the disease due to their presence in abundant numbers in the mouths of affected patients, the contagion theory was greatly reinforced.

Because of the "epidemic proportions" the disease assumed among the Allied forces in World War I when it was first recognized as a clinical entity, acute necrotizing ulcerative gingivitis came under much closer scrutiny of investigators. (Bowman (1916), Bouty (1917), Campbell and Dyas (1917), Goadby (1916), Laughlen, Warner and Holmes (1919), McKinstry (1917), Reckord and Baker (1920), Semple, Price-Jones and Digby (1919), Taylor and McKinstry (1917)). Many of these investigators assumed the disease to be highly contagious, and indicated case histories in support of this. However, the evidence in all cases was circumstantial, without any careful analysis of other etiologic factors. On the other hand, Semple, Price-Jones and Digby (1919) in their analysis of 3000 soldiers made no mention of contagion and felt that the control of the disease "rests with cleanliness of the mouth and skilled attention to the teeth".

Although repeated attempts were made to implicate the spirochete and fusiform bacillus by proponents of the contagion theory, (Kritschevsky and Seguin (1924), Pilot (1928), Pardee, Gordon and Riley (1928), Schroff and Bartels (1929), Belding and Belding (1932), Goodrow (1936), Cobe (1948), no one had been able to satisfy Koch's postulates of reproducing the disease in another host with the organisms. Schwartzman and Grossman (1941), using positive smears as their criteria for diagnosis, tried to transfer the infection from seven

A WORLD-WIDE SURVEY OF ACUTE NECROTIZING ULCERITIS (Trench Mouth) (Vincent's Infection)

NAME OF PATIENT	SEX M or F	RACE Cauc. Negro Asiatic	AGE	OCCUPATION	DEGREE OF SEVERITY M-Mild MM-Moderate S-Severe	DATE OF ONSET	DATE OF DIMISSAL	EVALUATION OF ORAL CLEANLINESS P - Poor F - Fair G - Good	NUTRITIONAL STATUS OF PATIENT P - Poor F - Fair G - Good	NO. OF PREVIOUS EXPERIENCES WITH DISEASE	ANY EVIDENCE OF CONTAGION IN CASE? Yes or No	CLASS OF I II III	WEATHER CONDITIONS ON PRESENTATION Rain Snow Fair

KEY I

1. L. calculus light
1. H. calculus heavy
2. Third molar flaps
3. Irregular position of teeth
4. Areas of food impaction
5. Local mechanical irritants
6. Tobacco
   p-pipe
   c-cigarette
   cr-cigar
   ch-chewing
7. Alcohol
8. Condiments

KEY II

A. Interproximal sloughing
B. Odor
C. Sudden onset
D. Spontaneous bleeding
E. Pain
F. Increased salivation
G. Metallic taste
H. Wedging sensation of the tooth
I. Anesthesia of periodontal membrane
J. Glossitis
K. Enlargement of the submaxillary and submental lymph nodes

G ULCE NITIS *(Trench Mouth)*
(Vincent's Infection)

DEPARTMENT OF PERIODONTIA AND ORAL MEDICINE
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NEW YORK UNIVERSITY COLLEGE OF DENTISTRY
209 EAST 23RD STREET
NEW YORK 10, NEW YORK, U.S.A.

CE IN	CLASS OF I II III	WEATHER CONDITIONS ON PRESENTATION Rain Snow Fair	METHOD OF TREATMENT (IF DRUGS USED, GIVE NAME (See Key III) List Letters, (a, b, c, etc.) of methods used	ANY CONCURRENT EMOTIONAL STRESS SITUATION FOR PATIENT? TYPE Yes or No Family, Work, School	OTHER ETIOLOGIC FACTORS (SEE KEY I) List numbers (1, 6c, etc.) of factors present	ENUMERATE DIAGNOSTIC SIGNS PRESENT IN CASE (SEE KEY II) List letters (A, B, C)	NAME OF CLINICIAN IN CHARGE OF CASE	ADDITIONAL COMMENTS

n of the
riodontal
the submaxil
ymph nodes

eneral symptom of toxemia
astro-intestinal symptoms
anthematous eruption
acteriological smears

KEY III

a. Scaling
b. Prophylaxis
c. Oxidizing agents
   e.g. H202, sodium perborate
d. Arsenical
e. Aniline dyes
f. Antibiotics
   e.g. sulfa, penicillin, aureomycin
g. Vitamins
h. Home care, mouthwash
i. Home rest
j. Soft diet

positive cases to eleven negative cases of intact gingivae and throat, and fourteen negative cases in which they mechanically traumatized the gingivae and throat. They were unsucessful in transmitting the disease in all 25 cases. King (1941) rubbed the material from an infected socket into an interdental papilla he had traumatized in his mouth. Although he had neglected his oral hygiene and Vitamin C intake for a few weeks previously, nothing occurred. Wilkie (1945), too, inoculated himself four times and did not get the disease. However, although acute necrotizing ulcerative gingivitis could not be reproduced in a new host, some investigators found that the organisms, injected into other parts of the body, e.g. lungs, were virulent enough to cause abcess lesions (Tunicliff and Hammond (1937), Smith (1927), Cobe, (1948).)

With the failure to experimentally reproduce the disease, the contagion theory lost many adherents, but the spirochete and fusiform bacillus continued to play a dominant role in the thinking of many investigators. Amies (1945) believed the organisms to be loosely related saprophytes which with ecological change become pathogenic. Belding and Belding (1932), however, claimed the organisms are initial invaders, always pathogenic but conceded that "susceptibility to infection depends primarily on lowered tissue resistance." Jewesbury (1943) stated they are opportunists rather than primary pathogens, while Kritschevsky and Seguin (1924) said the organisms are not initial invaders but the causative organisms, the severity of the disease being directly proportional to the number of organisms present. Pilot (1928), however, describes the organisms as normal inhabitants of the mouth, where an alteration in the symbiotic relationship causes an increase in the number of the organisms and simultaneously spreads the disease.

Even among the investigators who discount contagion, considerable thought has been given to the role of the organisms in the disease. Linton (1943) believes the organisms are seconday invaders in tissue debilitated by other causes, and Rosebury (1942) stated the organisms are harmless parasites associated with an endogenous infection, and that predisposing agencies which induce tissue damage permit undue proliferation. Shaffer (1953), however, in a study of thirty-six biopsies found that the bacteria invaded the tissue after it became necrotic rather than invading healthy tissue and causing necrosis. These latter views concur with Miller (1950) who believes that "the organisms may be saprophytes and as such find dying tissue or tissue with lowered resistance an ideal place for growth." He concludes that the bacteria are present because of the disease rather than the disease occuring because of the bacteria.

The Research Commission of the American Dental Association (1945) in an evaluation of this disease stated that although in the early days it had been thought to be highly infectious, "more recently, as a result of careful research, plus the observations and experiences of many clinicians in the field, this theory has been largely discounted."

Although it is now most widely accepted that acute necrotizing ulcerative gingivitis is not contagious, some investigators still maintain it is a communicable disease (Herzog (1951), Pindborg (1951)). For this reason, the column on contagion was included in the chart. Secondly, alleged cases of contagion which have appeared in the literature have had no real documentation. By enabling us to evaluate predisposing factors on the chart, e.g. oral hygiene, nutritional status, emotional background, etc., a more valid interpretation can be arrived at in cases where there may be a claim of contagious contact.

CLASSIFICATION OF OCCLUSION

There are no references in the literature as to a correlation between malocclusion and the incidence of acute necrotizing ulcerative gingivitis. It has been an observation of the authors and many investigators ((Miller (1950), Sorrin (1934)) that malocclusions and occlusal disharmonies may predispose an individual to periodontal conditions. It would be an interesting facet of data to determine whether any correlation, as we have postulated, does exist.

Angles' classification of occlusion was used only because the authors felt it is the most widely known, even though it is not as widely accepted.

SEASONAL VARIATIONS

Reckord and Baker (1920) reported that during 1916 while working at the Queens Alexandria Military Hospital, they noted the disease was most common in the winter months. In their records at Camp Devins Hospital from April 1918 to July 1919, the numbers increased in August (4), rose slowly in October and remained stationary to December (6) with a rapid rise in February (15), March (14) and then a decline to April (5), May (3) and June (2) cases. McKinstry (1917) too found the disease to be seasonal, and that it occurred mostly in the winter months. Webster (1941) also found winter months to be the season in which the incidence of the disease was greatest. Daley (1931) observed a higher incidence during the first three months of the year, and the lowest incidence in the last three months of the year. Miller and Greenhut (1944), in New York found the greatest occurrence of acute necrotizing ulcerative gingivitis during the month of October. Pindborg (1951) in Denmark, which is climactically similar to New York, also found August to October to have the greatest incidence of the condition. On the other hand, Wilkie (1945) in records he kept on 10,000 population from October 1941 to December 1942 found no seasonal variations. Carter and Ball (1953) in a three-year study at the Great Lakes Naval Dental Department found seasonal highs and lows but in the three years found no concurrence of these variations. Stammers (1944) noted an incidence-peak from November to March and then a leveling off, except for June, followed by another rise in September and October. He concludes "that there is a tendency to a seasonal variation—but that the variation is not of necessity constant—". Although these findings would seem to question a seasonal variation in the prevalence of the disease, it would still be particularly interesting to determine whether by recording climatic conditions a pattern of occurrence of the disease around the world might be revealed.

METHODS OF TREATMENT

Basically there are three schools of thought in methods of treating acute necrotizing ulcerative gingivitis; the method depending directly on the amount of emphasis one places on the role of the fusiform bacillus and spirochete in the etiology of the disease. One group of investigators who placed primary importance on the organisms has over a period of years empirically treated the disease with all sorts of medicaments, each man advocating his particular medication, each medication being different from those preceeding it. The result has been utter confusion, for literally over one hundred medicaments have been advocated. The second group divides the etiology between the organisms and oral hygiene. The advocates of this school of thought treat the "acute" phase with medications, and when the "painful, acute" symptoms disappear, they proceed with oral hygiene treatment, eliminating local irritational factors. The third group believes that the presence of the organisms in acute necrotizing ulcerative gingivitis is only a secondary manifestation overlaying a basic periodontal condition. This school discounts the use of any medication during the acute phase of the condition. They treat acute necrotizing ulcerative gingivitis as a chronic periodontal condition which has been precipitated into a acute episode by a superimposed stress situation such as overexertion, emotional problems, nutritional deficiencies, systemic disorders, or excessive local irritational factors.

Until recently, the chemotherapeutic aspects of the treatment of acute necrotizing ulcerative gingivitis received the greatest amount of attention. Investigators, in a zealous attempt to find a chemical that would be harmless to the oral mucosa yet effective against the spirochete and bacillus, have presented many medicaments. Unfortunately, most of the work with these medicinals has been empirical, with hardly any controlled scientific studies to determine best concentrations of the medicaments on their comparative efficacy. As a result, this has been one of the most controversial areas in dentistry.

1. *Oxidizing Agents.* This group of medi-

cinals release nascent oxygen, which supposedly has a bacteriostatic effect on the organisms associated with acute necrotizing ulcerative gingivitis. Hydrogen peroxide has been widely used by many investigators (Brooks and Wilson (1944); Bouty (1917) in the form of hot gargles; Farrell and McNichols (1937); Goodrow (1937); Jewesbury (1943); Stammers (1944)). On the other hand, some investigators have had poor results with its use (Carter and Ball (1953); Miller et al (1938); Rosenthal (1952); Schaffer (1954); Schuessler et al (1945).) Sodium perborate, although advocated by some practitioners, has been condemned by many investigators (Hirschfeld (1942); Miller et al (1938); Rosenthal (1952); Shallenberger et al (1945); Williams (1935)). Potassium permanganate, too, is not considered efficient by Rosenthal (1943).

2. *Arsenicals.* Because of the specificity of arsenicals for spirochetes, these medicinals received a great deal of attention. They were very popular during the first World War. Bowman (1916) employed a paste containing Fowler's solution and ipecacuanha wine. Bouty (1917) used arsphenamine intravenously, neoarsphenamine topically, and arsenic plus a mixture of sodium salicylate and potassium chlorate. Campbell and Dyas (1917) also used arsphenamine, neoarsphenamine and a liquor arsenicalis swabbed on the affected tissue three to four times daily. However, Stammers (1944) found the liquor arsenicalis ineffective. Linghorne and Stedman (1951) suggested an arsenate mouthwash. Farrell and McNichols (1937) recommended a topical application of neoarsphenamine plus glycerine 10 per cent, but were against intravenous neoarsphenamine. Jewesbury (1943), too, wrote strongly against intravenous use of arsenicals.

Although Goodrow (1937) and Rosenthal (1937, 1943, 1952) found arsphenamine effective, Brooks and Wilson (1944), Ludwick (1944) and MacGregor and Long (1945) advised against the use of arsenicals. Goodrow (1937) and Wihr (1946) reported good results obtained with neoarsphenamine, but Schaffer (1954) found it only fair and Schuessler et al (1945) reported it ineffective.

From these conflicting views, one can readily appreciate the controversy in the field.

3. *Dyes.* Although the use of antiseptic dyes in the treatment of acute necrotizing ulcerative gingivitis was very popular at one time, they are not employed widely now. Methylene blue used successfully by Bouty (1917) was found to give poor results by Farrell and McNichols (1937). They also found gentian violet unsuccessful in the treatment of acute necrotizing ulcerative gingivitis, but Miller (1950) suggested its use in a one per cent aqueous solution. Acriflavine is recommended by Miller (1950) and Stammers (1944); and Viogen (a one per cent aqueous solution of brilliant green and crystal violet) is suggested by Brooks and Wilson (1944); Miller (1950) and Wihr (1946).

4. *Caustics.* This group of chemicals, although they have a marked bactericidal effect on contact, produce tissue damage and deter the normal healing of the gingivae. Chromic acid, by far the most popular in this group has had many proponents (Brooks and Wilson (1944); Farrell and McNichols (1937); Goadby (1916); Goodrow (1937); Jewesbury (1943); Pardee et al (1928); Wihr (1946); Stammers (1944); and Shallenberger et al (1945)). However, it's use is not recommended by MacGregor and Long (1945), Rosenthal (1937) or Schuessler et al (1945). Silver nitrate is advocated by Bouty (1917) and Brooks and Wilson (1944); while copper sulfate and copper sulfate with mepharsen scored well in motility tests conducted by Rosenthal (1937), who felt they were not damaging to body tissues.

5. *Mercurials.* Miller (1950) and Rosenthal (1937) recommended the use of a 1:200 untinted tincture of metaphen. Goodrow (1937) successfully utilized mercuric chloride, Bouty (1917) found mercury perchloride and glycerin (1:500) effective and Wihr (1946) believed mercurochrome desirable. However, at the present time most mercurials are rarely used.

6. *Halogens.* The bactericidal effect of the halogens is produced by their ability to oxidize some essential components of bacterial protoplasm. Rosenthal (1952) found Dakin's solution effective. As a result of his motility tests

he also recommended Talbot's iodoglycerol (1937), 25 per cent aqueous solution of iodine (1943, 1952) and Lugol's solution (1937). However, Stammers (1944) found Lugol's solution ineffective. Wihr (1946) recommended Churchill's tincture of iodine but Carter and Ball (1953) found it poor. Bouty (1917) suggested 6 per cent tincture of iodine and Dillon (1940) advocated an iodine-glycerin-potassium iodide mixture. Box (1930) and Box and Ham (1942) and Miller (1950) suggest the Adams treatment which employs both Churchill's tincture of iodine and a 35 per cent silver nitrate solution. However, because of their toxicity to tissues and their irritant properties which deter healing, the halogens have been used less extensively.

7. *Antibiotics.* The use of sulfa drugs in local treatment of acute necrotizing ulcerative gingivitis was first suggested by Greenhut (1942). Linton (1943) and Manson and Craig (1945) found sulfathiazole tablets effective and Shallenberger et al (1945) obtained good results with sulfadiazine losenges.

During the second World War, with its stresses and tensions, and the concommitant rise in incidence of acute necrotizing ulcerative gingivitis, there was great interest evidenced in the disease. Penicillin, having become more available, received the greatest amount of attention. It was used in every conceivable manner and form.

It was employed as losenges by Glickman (1947), as troches by Hoerman (1948), as pastilles by MacGregor and Long (1945), Atkinson (1945); McIntyre (1945), Schaffer (1954), Schwartz (1945) and as tablets by Ziskin et al (1947). It was applied in solutions topically by Cofield et al (1945), Rosenthal (1945), Roxburgh (1945), Schuessler et al (1945), Shallenberger et al (1945) and Stewart (1946). It was applied in packs of 200,000 units by Stewart (1946) and 100,000 units by Thexton and Dick (1945). It was used in a barrier cream of gum tragacanth and penicillin by Christensen (1945) and a lanette wax with penicillin 1,000 units per cubic centimeter injected by syringe into interproximal spaces by Fido (1945).

Intramuscular injections of penicillin were recommended by many investigators (120,000 units by Christensen (1945); 20,000 units every 3 hours for 3 days and nights by Karensky and Strobin (1944), 25,000 units by Naegeli and Morginson (1945), 10,000 units every three hours for one to two days by Pearce and MacDonald (1945), 25,000 units every three hours by Schuessler et al (1945), and Shallenberger et al (1945), 1,250,000 units by Strock (1944), and 600,000 to 1,000,000 units by Sweeney et al (1945). Stern (1945) recommended local injection of 20,000 units daily for 3 days into the labial fold.

The effects of other antibiotics have also come under observation. Bacitracin was used by Kozol and Schuster (1952); penicillin and bacitracin troches by Carter and Ball (1953) and Rosebury (1946). Aureomycin was used orally by Goldman and Bloom (1949); and Grupe (1956); as troches by Heylings (1955) and Stewart and Roth (1950). Terramycin was employed by Carter and Ball (1953) and Shpuntoff and Shpuntoff (1954). Rosenthal (1950) found streptomycin effective.

Other agents have also been used. Zinc oxide and eugenol packs have been employed favorably by Box and Ham (1942), Cripps (1953), Fish (1938) Rosenthal (1943) and Stammers (1944).

Many investigators have used vitamin therapy effectively while others have found it of no value. These observations have been dealt with in the nutritional status section.

The components of Key III are just a few of the various methods of treatment that are in use today. It is hoped that the clinicians will be as detailed as possible in noting all facets of treatment employed with each patient. In this way, it may be possible to cull some basic trends in the treatment of acute necrotizing ulcerative gingivitis.

EMOTIONAL STRESS

It has been the contention of some investigators that emotional or physical stress situations may precipitate a chronic periodontal condition into an acute episode. (Miller and Firestone (1947) and Roth and Stone (1951)).

Stammers (1944) observed that the first great increase in the number of cases of acute necrotizing ulcerative gingivitis seen was when the city "was experiencing periodical heavy air-raids, and almost nightly 'Alerts'". Kerr (1945) found a higher incidence of the disease around exam time. Schluger (1949) reported a rise in the occurrence of acute necrotizing ulcerative gingivitis with fatigue and trauma; and Hester et al (1952) stated that there were about 5,000 exacerbations as a result of long flights at low altitudes, or short flights at high altitudes.

From these observations, it would seem that many instances of emotional or physical stress factors may have been overlooked because investigators did not attach much importance to them. Since the patients will be questioned about emotional and physical stress situations in this study, some interesting facets may be revealed.

OTHER ETIOLOGIC FACTORS

An enumeration of some etiologic factors not covered in other columns of the chart are listed in Key I. Third molar flaps are the cause of 80 per cent of cases observed according to McIntyre (1945). Brooks and Wilson (1944) also felt that many cases they had seen had started in the soft tissue around the third molar area.

Carter and Ball (1953) observed overindulgence in drinking or smoking in 48 per cent of the cases studied in a three year survey. Kerr (1945), because most patients noticed symptoms on Tuesday and Wednesday, felt that the occurrence of acute necrotizing ulcerative gingivitis in their cases was due to excessive use of tobacco and alcohol over the weekend. Pardee et al (1928) felt that smoking probably delayed the cure they were able to effect and Stammers (1944) found that a large number of his patients were very heavy smokers.

The clinician, by indicating which etiologic factors are operative in each case, will give us a tool in evaluating the importance of the various factors involved.

The diagnostic symptoms that most investigators employ in making a diagnosis, are listed in Key II. It is hoped that the clinician will indicate all the symptoms he observes in the patient on presentation which lead him to a diagnosis of acute necrotizing ulcerative gingivitis.

* * * *

We are planning to send a copy of this chart to every dental school in the world. Translations of the chart have been made into French, German, Italian, Portuguese and Castillian to aid clinicians in recording the data efficiently. It is planned to have this chart hung in that clinic of each dental school where patients suffering with acute necrotizing ulcerative gingivitis would appear for diagnosis and treatment. In this way the information and history elicited from the patient could be recorded onto the chart immediately. Each year, for five years, fresh charts will be sent to each school, and the previous year's completed charts should be returned to us for preliminary compilation and evaluation of the data. We have arbitrarily chosen five years for the duration of the study so that any statistical irregularities (e.g. seasonal variations) would be cancelled out unless they significantly recurred during the five years. This, plus the greater number of cases that would be available for study over a longer period of time entered into our decision for a five-year study. The study is to officially begin on September 1, 1958 and continue until August 31, 1963.

Below are listed all of the schools that have been invited to participate in this study. This list is much larger than the number of schools contacted in our preliminary study. We wish to acknowledge, however, by means of an asterisk, our gratefulness to those schools that cooperated so completely in our first study.

AFRICA

University de Dakar, Dakar, Fr. West Africa
Alexandria University, Alexandria, Egypt
*Cairo University, Gidza, Cairo, Egypt
*University of the Witwatersrand, Johannesburg (Transvaal), South Africa
University van Pretoria, Pretoria (Transvaal), South Africa

ASIA

University of Ceylon, Peradeniya, Ceylon
National Defense Medical Center, Taipei, Taiwan, China
Gadjah Mada University, Jogjakarta (Java), Indonesia
Universite de Teheran, Teheran, Iran
Royal College of Medicine, Baghdad, Iraq
Seoul National University, Seoul, Korea
L'Universite Saint-Joseph, Beyrouth, Lebanon
University of Malaya, Singapore, Malaya
DeMontmorency College of Dentistry, Lahore, Panjab, Pakistan
Universite Syrienne, Damas, Syrie
University of Medicine, Bangkok, Thailand
Universite Nationale du Viet Nam, Saigon, Viet Nam
Hebrew University Dental School, Jerusalem, Israel

India

Sir Currimbhoy Ebrahim Memorial Dental College, Bombay, India
The Nair Hospital Dental College, Bombay
*Calcutta Dental College, Calcutta
King George's Medical College, Lucknow (Uttar Pradesh)
Dental College, Solan (East Panjab)

Japan

Kyushu University, Fukuoka
Osaka Daigaku, Osaka
Nihon University, Tokyo
Tokyo Ika Shika Daigaku, Tokyo
Mie Prefectural University, Tu-Si (Mie-Ken)
Yamaguchi Medical School, (Yamaguchi Prefectural)
Yokohama University School of Medicine, Yokohama

Australia, New Zealand, Philippines

University of Adelaide, Adelaide, Australia
University of Queensland, Brisbane, Australia
University of Melbourne, Melbourne (Victoria), Australia
*The University of Western Australia, Nedlands, Australia
University of Sydney, Sydney (New South Wales), Australia
*The Australian College of Dentistry, Melbourne (Victoria), Australia
*University of Otago, Dunedin, New Zealand
University of New Zealand, Wellington, New Zealand
University of the East, Manila, Philippines
Manila Central University, Manila, Philippines
National University, Manila, Philippines
Centro Escolar University, Manila, Philippines
Central Philippine University, Iloilo City, Philippines
University of the Philippines, Quezon City, Manila, Philippines

EUROPE

France

*Universite de Paris a la Sorbonne, Paris
Universite de Bordeaux, Bordeaux
Universite de Nancy, Nancy
University de Toulouse, Toulouse
Universite de Poitiers, Poitiers
Ecole Dentaire de Paris, Paris
Ecole Dentaire Francais, Paris
Ecolde Chirurgie Dentaire et de Stomatologie de Paris, Paris
Ecole Dentaire, Paris

Germany

Freie Universität Berlin, Berlin
Rheinsche Friedrich - Wilhelms - Universität Bonn, Bonn
Univesität Köln, Köln
Friedrich-Alexander Universität, Erlangen
Johann Wolfgang Goethe Universität, Frankfurt-on-Main
*Albert-Ludwigs-Universität, Frieburg
*Georg-August-Universität Zu Göttingen, Göttingen
Universtät Griefswald, Griefswald
Universität Hamburg, Hamburg
Ruprechs-Karl-Universität, Heidelberg
Christian-Albrechts-Universität, Kiel
Karl-Marx-Universität, Leipzig, Leipzig
Johannes-Gutenberg-Universität, Mainz
Philipps-Universität, Marburg
Ludwig-Maximilians-Universität München, Munich
Universität Münster, Münster
Universität Rostock, Rostock
Eberhard-Karls Universität, Tübingen

Great Britain

*Birmingham University, Birmingham
Bristol University, Bristol
Edinburgh University, Edinburgh, Scotland
Glasgow University, Glasgow, Scotland
University of Leeds, Leeds
University of Liverpool, Liverpool
University of London, London
*Guy's Hospital Dental School, London
*Royal Dental Hospital of London, London
Institute of Dental Surgery, Eastman Dental Hospital, London
Victoria University of Manchester, Manchester
*St. Andrews University, St. Andrews, Fife, Scotland
*Sutherland Dental School, University of Durham, Newcastle upon Tyne
Senior Medical and Dental School, Dundee, Scotland
*University of Sheffield, Sheffield

Italy

*Medico Chirurgo Odontoiatra, Messina
Universita Degli Studi, Pisa
Universita Degli Studi, Rome
Universita Degli Studi, Sassari, Sardinia
*Instituto Superiore di Odontoiatra, Rome

Leopold-Franzens Universität, Innsbruck, Austria
Rijksuniversitet Te Gent, Gent, Belgium
Universite De Liege, Liege, Belgium
Universite Catholique De Louvain, Louvain, Belgium
Karlova Universita, Prague, Czechoslovakia
Frantisek Palacky Universita, Olomouc, Czechoslovakia
*Aarhus Universitet, Kobnhavn, Denmark

Loràn Eötvös Tudomànyegytem, Budapest, Hungary
Szegedi Orvostdomànyi Egyetem, Szeged, Hungary
Pècsi Tudomànyegyetem, Pècs, Hungary
*University College, Dublin, Ireland
*Queen's University of Belfast, Belfast, Ireland
Rijksuniversiteit Te Utrecht, Utrecht, The Netherlands
Rijksuniversiteit Te Groningen, Groningen, The Netherlands
Norges Tannlegehogskole, Geitmyrsveien, Oslo, Norway

Poland

Uniwersytet Jagiellonski, Cracow
Uniwersytet Lòkzki, Lòdz
Uniwersytet Poznanski, Poznan
Uniwersytet I Politechnika We Wrocawiv, Wrocaw
Akademia Stomatologiczna, Warsaw

Universidad De Madrid, Madrid, Spain
Tanläkarhögskolan: Malmö, Malmö, Sweden
Tandläkarhögskolan i Stockholm, Stockholm, Sweden
Universitat Bern, Bern, Switzerland
*Universite De Geneve, Geneva, Switzerland
Institut De Medecine Dentaire, Geneva, Switzerland
State University of Damascus, Damascus, Syria
Istanbul Universitesi, Istanbul, Turkey
Universität Zürich, Zürich, Switzerland

U.S.S.R.

Kazan Stomatological Institute, Kazan, Tatar
Kharkov Stomatological Institute, Kharkov
Kiev Stomatological Institute, Kiev
Leningrad Stomatological Institute, Leningrad
Molotov Stomatological Institute, Molotov
Moscow Stomatological Institute, Moscow
Tbilisi Stomatological Institute, Tbilisi

Yugoslavia

University of Zagreb, Zagreb
Belgrade School of Medicine, Belgrade
Medicinska Visoka Sola v Ljubljani, Ljublani
Advanced School for Dentists, Sarajevo

SOUTH AMERICA

Argentina

Universidad de Buenos Aires, Buenos Aires
Universidad Nacional de Córdoba, Córdoba
Universidad Nacional del Litoral, Santa Fé

Bolivia

Universidad Mayor de San Simón, Cochabamba
Universidad Mayor de San Andrés, La Paz
Universidad Mayor, Real Y Pontificia de San Francisco Xavier de Chuquisaca, Sucre

Brazil

Faculdade de Odontología do Pará, Belém (Pará)
Universidade de Minas Gerais, Belo Horizonte (Minas Gerais)
Faculade de Odontología de Campinas, Campinas (São Paulo)
Universidade do Parana, Curitiba (Parana)
Faculade de Farmácia e Odontología de Santa Catarina, Florianopolis (Santa Catarina)
Faculade de Farmácia e Odontología do Ceara, Fortaleza (Ceara)
Escola de Farmácia e Odontología de Alfenas, Alfenas
Faculdade de Farmácia e Odontología de Araraquara, Araraquara (São Paulo)
Faculdade de Farmácia e Odontología de Goias, Goiania (Goias)
Faculdade de Odontología da Paraiba, João Pessoa (Paraiba)
Faculdade de Farmácia e Odontología de Juiz de Fora, Juiz de Fora (Minas Geraia)
Faculdade de Farmácia e Odontología de Natal, Natal
Faculdade de Farmácia e Odontología do Estado do Rio de Janiero, Niterói (Rio de Janiero)
Universidade do Rio Grande do Sul, Porto Alegre, (Rio Grande do Sul)
Universidade do Recife, Recife (Pernambuco)
*Universidade do Brazil, Rio de Janeiro (Distrito Federal)
Faculade de Farmácia e Odontología de São Luiz São Luiz (Rio Grande do Sul)
Universidade de São Paulo, São Paulo
Faculdade de Farmácia e Odontología de Ribeirao Preto, Ribeirao Preto (São Paulo)
Universidade do Rio Grande do Sul, Pelotas (Rio Grande do Sul)
Faculdade de Odontología do Triangulo Mineiro, Uberaba (Minas Gerais)
Faculdade de Odontología do Espirito Santo, Vitoria (Espirito Santo)

Universidad de Concepción, Concepción, Chile
*Universidad de Chile, Santiago de Chile, Chile
*Universidad Nacional de Colombia, Bogotá (Cundinamarca), Colombia
Pontificia Universidad Catolica Javeriana, Bogotá, Colombia
Universidad de Cartagena, Cartagena (Bolivar), Colombia
*Universidad de Antioquia, Medellin (Antioquia), Colombia
Universidad Nacional de San Jose de Costa Rica, San Jose, Costa Rica
Universidad de la Habana, La Habana, Cuba
Universidad de Cuenca, Cuenca (Azuay), Ecuador
Universidad Central del Ecuador, Quito (Pichincha), Ecuador
Universidad de San Carlos de Guatemala, Guatemala, Guatemala
Universidad Nacional de Nicaragua, Leon, Nicaragua
Universidad de Guayaquil, Guayaquil, Guayas, Ecuador
Universidad Nacional del Paraguay, Asuncion, Paraguay
Universidad Nacional Mayor de San Marcos de Lima, Lima, Peru
Universidad Nacional Autonoma de Av. Cuscatian y 2a Calle Poniente, San Salvador, El Salvador
*Universidad de La Republica, Montevideo, Uruguay
Universidad Central de Venezuela, Caracas (Distrito Federal), Venezuela

Universidad Nacional del Zulia, Maracaibo (Zulia), Venezuela
Universidad de Los Andes, Merida (Estado Merida), Venezuela
Universidad de Santo Domingo, Republica Dominicana
Universite D'Haiti, Port-au-Prince, Haiti
Universidad de Honduras, Tegucigalpa, Honduras

NORTH AMERICA

Mexico

Universidad de Guadalajara, Guadalajara
Universidad Autonoma de Guadalajara, Guadalajara
Universidad de Yucatan, Merida (Yucatan)
Universidad Nacional Autonoma de Mexico, Mexico (Distrito Federal)
Universidad de Puebla, Puebla (Puebla)
Universidad Autonoma de San Luis Potosi, San Luis Potosi
Universidad de Nuevo León, Monterrey (Nuevo León)

Canada

University of Alberta, Edmonton (Alberta)
Universite de Montreal, Montreal (Quebec)
McGill University, Montreal
University of Toronto, Toronto (Ontario)
Dalhousie University, Halifax, Novia Scotia

United States

School of Dentistry, University of Alabama, Birmingham, Alabama
*School of Dentistry, College of Physicians and Surgeons, San Francisco, California
*College of Dentistry, University of California, San Francisco, California
School of Dentistry, University of Southern California, Los Angeles, California
School of Dentistry, College of Medical Evangelists, Loma Linda, California
*School of Dentistry, Georgetown University, Washington, D. C.
College of Dentistry, Howard University, Washington, D. C.
School of Dentistry, Emory University, Atlanta, Georgia
*Chicago College of Dental Surgery, Loyola University, Chicago, Illinois
The Dental School, Northwestern University, Chicago, Illinois
College of Dentistry, University of Illinois, Chicago, Illinois
*School of Dentistry, Indiana University, Indianapolis, Indiana
College of Dentistry, The State University of Iowa, Iowa City, Iowa
*School of Dentistry, University of Louisville, Louisville, Kentucky
School of Dentistry, Loyola University, New Orleans, Louisiana
Baltimore College of Dental Surgery, University of Maryland, Baltimore, Md.
Harvard School of Dental Medicine, Boston, Massachusetts
Tufts University School of Dental Medicine, Boston, Massachusetts
School of Dentistry, University of Detroit, Detroit, Michigan
School of Dentistry, University of Michigan, Ann Arbor, Michigan
*School of Dentistry, University of Minnesota, Minneapolis, Minnesota
*Kansas City-Western Dental College, The University of Kansas City, Kansas City, Missouri
School of Dentistry, St. Louis University, St. Louis, Missouri
*School of Dentistry, Washington University, St. Louis, Missouri
School of Dentistry, The Creighton University, Omaha, Nebraska
*College of Dentistry, University of Nebraska, Lincoln, Nebraska
The Dental School, Fairleigh Dickinson University, Teaneck, New Jersey
College of Dentistry, Seton Hall University, Jersey City, New Jersey
School of Dental and Oral Surgery, Columbia University, New York, New York
College of Dentistry, New York University, New York, New York
*School of Dentistry, University of Buffalo, Buffalo, New York
School of Dentistry, The Ohio State University, Columbus, Ohio
School of Dentistry, Western Reserve University, Cleveland, Ohio
The Dental School of the University of Oregon, Portland, Oregon
*School of Dentistry, Temple University, Philadelphia, Pennsylvania
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Needless to say, the validity and significance of this study will depend completely on the cooperation of all the clinicians and schools involved. We can only hope that each man's desire to further the science of dentistry will act as a spur.

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UN STUDIO DE STATISTICA MUNDIAL IN RE ACUTE GINGIVITIS ULCERATIVE NECROTISANTE

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SUMMARIO IN INTERLINGUA

Le effortios de trovar le factores etiologic e un tractamento definitive pro acute gingivitis ulcerative necrotisante abunda in controversias. Le etiologia del morbo ha essite attribuite a multe differente factores—factores de contagion, saisonal, psychosomatic, bacterial, nutrimental, etc.—e le methodos proponite pro su tractamento e medication ha essite phantasticamente numerose. Iste tremende varietate de opiniones ha resultate in un confusion que de su parte obscura le areas de accordo in que conceptos rational poterea esser postulate.

Iste rationes ha stimulate le decision de initiar un definitive studio statistic, le datos del qual servirea como fundamento de recercas basic in le campo in question. Nostre prime essayo esseva inviar un detaliate questionario a varie scholas medical in omne partes del mundo. Tamen, nos ha trovate que (1) multe scholas habeva incomplete fundos de documentation in consequentia del guerra e (2) omne schola seque su systema individual in le registration del datos, de maniera que nos non poteva obtener le information necessari pro formular conclusiones valide.

A causa de iste insuccesso in le effortio de obtener datos digne de confidentia ab le archivos de notationes passate, le decision esseva facite de organisar un studio in que nove datos esserea colligite per medio de formularios standardisate. Le formulario esseva elaborate con le objectivos de (1) coperir omne le aspectos controverse ab le punctos de vista etiologic e therapeutic e (2) colliger datos capace forsan a revelar predilectiones del morbo de un typo statisticamente interessante. Un detaliate discussion del principios rational—derivate ab le studio del litteratura—pro le selection del varie componentes del formulàrio representa le corpore del presente articulo.

Nos plana inviar exemplares del formulario a omne schola dental in le mundo. Traductiones de illo ha essite preparate in francese, germano, italiano, portugese, e castiliano pro assister le clinicos in le efficace registration del datos. Le intention es placiar le formulario intra omne schola dental in le clinica ubi patientes qui suffre de acute gingivitis ulcerative necrotisante se presenta pro diagnose e tractamento. In iste maniera il va esser possibile notar immediatemente in le formulario omne le informationes obtenite ab le patiente relative al historia de su caso. Omne anno—durante un periodo de cinque annos—nove formularios va esser inviate al scholas dental, e le completate formularios del anno precedente va esser retornate a nos pro un compilation e evalutation preliminari del datos. Le comenciamento official del studio va esser le 1 de septembre 1958. Illo va continuar usque al 31 de augusto 1963.

Un lista de omne le scholas invitate a participar in iste studio es presentate. Il va sin dicer que le validitate e le signification del studio va depender integremente del cooperation de omne le clinicos e de omne le scholas concernite. Nos pote sperar que le desiro de omne individuo de promover le scientia del dentisteria va ager como un stimulo.

DENTAL RESIDENCY PROGRAM IN ORAL MEDICINE†

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Editor's Note: This report is the program which the American Academy of Dental Medicine fostered. The New York University College of Dentistry and the Montefiore Hospital established this Residency in Oral Medicine.

The dental residency program recently completed by Dr. Sheldon Ross proved to be another milestone for both the American Academy of Dental Medicine and the dental department of Montefiore Hospital. Many hours were spent before the program could be properly planned and executed. It required the combined efforts of those educators of both Montefiore Hospital and the New York University College of Dentistry who collaborated to develop and establish this broad, liberal and far reaching program. The following men have been responsible for the development and success of the program: Dr. Raymond J. Nagle, Dean of the New York University College of Dentistry; Dr. Martin Cherkasky, Medical Director of Montefiore Hospital; Dr. David Tanchester, Chief of the Dental Service; Dr. Samuel Charles Miller, Professor and Chairman of the Department of Periodontia and Oral Medicine, New York University College of Dentistry; Dr. Sidney Sorrin, Associate Professor of Periodontia and Oral Medicine, New York University College of Dentistry, Periodontist at Montefiore Hospital and representing The American Academy of Dental Medicine.

The dental resident in oral medicine, which is a straight residency, enjoys the same privileges, accommodations and prerequisites provided by the hospital for residents in other services. The hospital issues to the dental resident a certificate of accomplishment upon the completion of his residency.

Among the training programs available for the preparation of candidates for specialty board examination are the approved internships and residencies listed by the Council on Dental Education. It is the general policy of the recognized dental specialty boards to recognize training which a candidate has received in a straight internship or residency as acceptable toward the satisfaction of the educational requirements of the boards.

The average life expectancy for the human has been greatly extended. Disease processes to which the individual is subject, are rapidly being brought under control, and many conditions which formerly may have terminated fatally are now wholly or partially conquerable. There is a trend for short term hospitalization; to return the patient back into society as soon as possible and to rely more on home care rather than on the medical facilities of institutions. As a result of these factors, there is an increasingly large number of people with complicated systemic backgrounds, who will be searching for dental care at the office of the private dental practitioner. The dentist will face the acute need for special ability over and above the training afforded by the dental schools today. Patients who are aware of their medical history will have to rely on the judgment of the dentist as to the limitations of treatment. Likewise, patients who are ill, but unaware of the fact, must depend upon the dentist to refer them to the proper medical specialist for adequate diagnosis and treatment. To supply the training and experience along these lines, a Residency Program in Oral Medicine is necessary; and a Residency Program to be successful, requires the facilities of a *modern dental school and a teaching hospital.*

If an arbitrary schedule of 48 units were to be outlined according to the U.S. Army Resi-

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† We are grateful to Dr. Sheldon Ross for much of the information contained in this article.

dency Program, the accompanying time distribution is observed:

1. Attendance at a course in advanced dental specialty ..... 16 units
2. Additional study in related subjects ..... 5 units
3. Technical laboratory assignments related to specialty ..... 6 units
4. Conferences, lectures, seminars, interne supervision ..... 4 units
5. Clinical experience ..... 17 units

This distribution may be adequate for a mechanical phase of restorative dentistry, but will not comprehensively allow for the broader, non-technical phases of oral medicine. The most marked changes would have to occur in phase 1, the attendance at a course in advanced dental specialty, since all four phases which follow are part of phase one as related to Oral Medicine.

The Council of Dental Education of the American Dental Association concerned with such a program recognizes a Straight Residency only, as opposed to the rotating or mixed type program offered to Dental Internes. With this in mind, the outline for the program in Oral Medicine should consist of those topics directly involved in the practice of Oral Medicine. This would allow for some redirection of emphasis on the allied subjects, to be determined by the facilities which the hospital has to offer.

RESIDENCY PROGRAM

A. Basic Science Review; Anatomy, Histology, Physiology, Biochemistry, Pharmacology, Bacteriology, Pathology.

B. Dental Services; Periodontia, Temporomandibular studies, Surgery, Anaesthesiology, Oro-facial Prosthesis, Roentgenology and Diagnosis.

C. Affiliation with Hospital Services
   1. Internal Medicine; including Metabolic, Endocrine, Neoplastic, Pulmonary, Cardiac, Dermatology Services.
   2. Head and Neck Service; including Radiology, Roentgenology, Surgery, Plastic Surgery, E.N.T.
   3. Pathology; Gross, Micro, and Clinical Laboratory Services.

D. Short Teaching Program—to encompass all above mentioned hospital services in outline.

E. Rounds and Conferences of Dental Services.

F. Rounds and Conferences of Medical Services.

G. Interne Teaching; including Distribution of work, consultation on cases, preparation of case reports.

The details of the material covered in the Residency Program follow below, with specific topic notations.

BASIC SCIENCE

(Each session is given under the direct supervision of the Chairman of the respective departments.)

HISTOLOGY—6 sessions, 2 hours each.

1. Review of Fundamental Tissues.
2. Development, calcification, metabolism, growth, structure of bone.
3. Development of tooth to stage of occlusion.
4. Development, structure, vitality and age changes of dentin, enamel, and cementum.
5. Eruption of teeth; mucous membranes; formation and structure of gingiva and periodontal membrane. Age and retrogressive changes.
6. Pulp-development; structural relations in deciduous and permanent teeth — retrogressive changes.

BACTERIOLOGY—9 sessions, 2 hours each.

1. Infection and Resistance.
2. Hypersensitivity and Immunity.
3. Ecology of the mouth — Environmental factors and flora.
4. Dental caries — Theories and Process, Dental caries — Factors in Etiology.
5. Dental caries — Tests, Prevention, and Control.
6. Sterilization in Dentistry; Cavity, Tooth, Tissues, Instrument and office routine.
7. Stomatitis; Infective (bacterial, viral, fungal).
8. Microbiology in Endodontia; Microbiology in Oral Surgery.
9. Focal Infection.

PATHOLOGY—21 sessions, 3 hours each.

1. Circulatory disturbances, blood coagulation, hemorrhage; blood dyscrasia-bleeding disease, hemophilia, thrombosis; coronary artery thrombosis, infarction.
2. Venous thrombosis; Embolism, infarction, necrosis; Healing and repair. Inflammation—Instruction—types of Inflammation.
3. Inflammation
   a) cell study—peripheral blood and bone marrow.
   b) reticulo—endothelial system.
   c) endocarditis—acute, subacute, chronic.
4. Inflammation; acute-abscess; osteomyelitis; upper lip infection-acute; appendicitis; healing-repair; R.E. system.
5. Inflammation, Tuberculosis, Syphilis.
6. Caries of Enamel.
7. Periodontal Diseases.
8. Acute Oral Infections; Alveolar abscess; Osteomyelitis; chronic periapical infection.
9. Chronic periapical infection; Fracture; Healing socket.
10. Diseases of the Blood.
11. Laboratory procedures; Mucous membrane lesions.
12. Vitamins; Endocrines.
13. Mucous membrane lesions.
14. Endocrines; Lymph node diseases.
15. Tuberculosis of oral cavity.
16. Death in Dentistry; Foci of infection.
17. Tumors. (General)
18. Tumors. (Specific)
19. Oral Tumors.
20. Odontogenic Tumors.
21. Tumors of bone; Fibrous dysplasia of bone.

ANATOMY—6 sessions, 3 hours each, on cadaver.

1. The skull-landmarks and important structures, the distribution of the Trigeminal Nerve.
2. The Cranial Cavity, cranial nerves, the plan of the nervous system; Some Reflexes encountered in Dentistry.
3. The Face.
4. The Pterygomandibular space, the Temporomandibular joint.
5. The maxilla, the nasal cavity, distribution of nerves and vessels to the upper jaw.
6. The floor of the mouth, the neck, the digastric triangle.

PHYSIOLOGY—8 sessions, 1 hour each.

The primary purpose of the work in physiology is to provide the scientific background necessary for subsequent diagnosis and treatment of the patient in terms of all the bodily functions involved in dental health and disease. The courses in physiology, biochemistry, and anatomy have been so coordinated as to provide an integrated treatment of topics covered; they follow a dovetailed schedule that affords mutual reinforcement without needless duplication.

Relations of physiology and biophysics to anatomy, biochemistry, and clinical dentistry. The concept of homeostasis. The basic unit of physiological function. The internal environment. Basic norms and limits of physiological reaction. The nature of sensory stimulation.

The neuromuscular unit. The biophysical properties of striated, cardiac, and smooth muscle. The nature of the nerve impulse and the properties of nerve. Action potentials. Effect of anesthetic and physical agents on nerve conduction. Pulp testers. The simple reflex arc.

Neural integration. Functions of the medulla, cerebellum, thalamus, hypothalamus, and cerebral cortex. Modern concepts of cerebral localization. Functions of sensory and motor pathways. The pyramidal and extrapyramidal systems. Control of posture. Flexor and extensor patterns in the spinal cord. The autonomic nervous system. Human reflexes. Electroencephalography. Epilepsy and neuropathies. Referred pain and neuralgias. Dental pain. The special senses.

Circulation. Haemodynamics, cardiac cycle, nervous and chemical control of heart rate and blood vessels. Arterial, venous, and capillary pressure. Circulation of the gingivae and dental pulp. Hemorrhage and blood clotting. Shock, cardiac failure. Circulatory reactions in general anesthesia.

Respiration. Mechanics of respiration, respirometry, the nervous and chemical control of respiration, the oxygen and carbon dioxide transport. Anoxemia, asphyxia. Respiratory reactions in general anesthesia.

Digestion. Peristalsis, control of sphincters, secretion, absorption, vitamins and dental health.

Metabolism. The basal metabolism. Cellular respiration. Enzymology.

Body fluids. Fluid compartments of the body, fluid exchange, formulation of interstitial fluid. The salt balance. Excretion by the kidney. The maintenance of the acid base balance.

Endocrines. Functions of the glands of internal secretion and their inter-relationships. Dental aspects of endocrine function. Allergic manifestations in the mouth.

Growth, degeneration, and regeneration. General principles, bone growth, alveolar bone, the "bone factor," physiological basis of orthodontics, functions of the periodontal membrane, wound healing, physiology and oral surgery, physiology and periodontia.

BIOCHEMISTRY—8 sessions, 1 hour each,

A. Fundamental Theories of Chemical Reactions.
   1) Carbohydrate.
   2) Lipids.
   3) Proteins.
      a) Protein proper.
      b) Amino acids.
   4) Vitamins and Hormones.
   5) Nucleoproteins.
   6) Digestion.
   7) Enzymes—general considerations including:—
      a) Composition.
      b) Specific activity.

B. Metabolism.
   1) Carbohydrate.
   2) Lipids.
   3) Protein including amino acids.
   4) Minerals.
   5) Water including urinary studies.
   6) Blood.
      a) Composition, Properties and Functions.
      b) Clinical analysis and tests.
      c) Clotting mechanism.
   7) Respiration.
   8) Nutrition.

PHARMACOLOGY—2 sessions, 2 hours each.

1. Prescription writing; Idiosyncracy and drug hypersensitivity; premedications, Analgesics.
2. Hemostatics; Local Anesthetics: Antibiotics.

Of the available series of Rounds, Lectures, Seminars, Conferences, Meetings, and Grand Rounds, numbering some 80 odd sessions, the following offer the most material related to oral medicine:

1, 2) Gross Pathology conferences twice weekly, 3) Clinical Pathology Conference, 4) Histopathology Conference, 5) Head and Neck Service Conference and Grand Rounds, 6) Medical Dept. Seminar, 7) Grand Rounds—Neoplastic and Metabolic, 8) Dental Dept. Conference.

While these eight officially scheduled sessions represent only about 9% of the total formally scheduled material scheduled material available, much more time is usefully applied in:—

a) Making rounds on all services with the internes.

b) Attending individual seminars or conferences on a non-regular basis, but rather as individual cases of interest arose and were followed up.

c) Making rounds on services as part of observations for research projects.

d) Consultation with other departments; the dental resident is required to make an oral diagnosis.

e) Cooperation with other departments to aid them in diagnosis and/or treatment related to the head and neck.

f) Attending a series of orientation lectures for the house staff which covered laboratory procedures, social medicine, cardiac emergencies, ENT emergencies, five seminar-lectures on hematology, etc.

g) Treatment of house-staff of other services affording opportunity to discuss cases, oral manifestations, etc. and permitting the dental resident to acquaint the medical staff with the aims of such a program, and to instruct the medical staff in fundamental dental therapy, procedures, and practices. It also permits frank, complete discussion of the limitations to be imposed on dental care for particular types of patients, and precautions to be followed to avoid fatalities related to dentistry.

h) Instruction of the dental internes in case reporting, teaching on the clinic floor, and preparation of regular dental staff meeting programs.

i) Research projects.

j) Treating patients in the dental clinic. Each patient reports to the clinic with his hospital chart. All information is available to the dentist. (Montefiore hospital is unique in that it is one of the few hospitals where the dentists' status is not "sub rosa.")

All the above ten methods for gathering information and material serve to bring the resident into contact with many other services—such as with Pulmonary; with Orthopedic for making mouth props; with Radiotherapy for designing shields for lips and tongue etc., in radon implants; with Radiotherapy for extractions before cobalt or x-ray therapy; with Metabolic for discussions of nutrition, calcification etc.; with Neurology on reflexes, paralysis, etc.; with Cardiology in reference to hypertensives and so on ad infinitum. It would take an entire volume to enumerate all the material available and the contacts made during the training period of a year.

In reference to the formal group of conferences mentioned before, it would be wise to point out that although the titles refer to one service, the conferences always have representatives from allied groups for discussion and presentation.

The Neoplastic and Metabolic Grand Rounds are a most informative series of conferences at which several patients are presented daily. The purpose of the meeting is to evaluate, diagnose and determine management of the patients who are presented. The house staff on this particular service presents the patients' history, laboratory findings, complaints etc., and the physician's findings in preliminary examination. The patient is then demonstrated so that all pertinent signs and symptoms can be shown. Everyone present may then examine the patient after which the patient is removed and an attending physician discusses the case. The differential diagnosis is presented, and then an elaboration on the patient's disease follows. Attendings from other services present the conclusions that their own services have come to at meetings held previously, and specialists in the field comment on the disease in general, and on the particular patient specifically. One reason that this conference is so valuable is that most metabolic diseases frequently show oral manifestations. Many are concerned with bone, blood and soft tissue repair, and the mouth and dental apparatus are prone to reflect early changes in these structures.

The gross pathology conferences are meetings of the Pathology Department to which the other medical specialties send representatives interested in a particular patient who has succumbed. It approaches the ideal way to teach medicine. History, lab findings, observations, etc. are all read to the group. Diagnostic Roentgenology presents all available x-ray series for interpretation. Interested persons are then questioned as to rationale for the procedures followed. After this, all the autopsy material is shown and the *clinical diagnoses* are confirmed or altered and blocks are prepared of all demonstrable lesions for sectioning and staining. It is to the credit of the Chief of the Pathology Service that he relentlessly pushes for evidence to account for every sign, complaint, or observation made on the patient.

The histopathology sessions during the week carry this one step further—in that the slides prepared from the blocks taken at gross conference are shown, and the tissue evidence is used to arrive at a final diagnosis at which time the case is signed out.

The Clinical Pathology Conference is the

windup of the above mentioned three weekly conferences. At this session the entire house staff and the entire attending staff meet and are given printed protocols detailing the patient's history, findings, observations, course in the hospital, etc. An Attending, previously in charge of the case, presents his clinical diagnosis. X-rays are presented, and the pathology department then demonstrates by kodachromes and microscopic slides the actual findings, the errors, and the misconceptions that led to the demise of the patient. In many cases the sessions are used to confirm correct medical diagnosis where the material is especially interesting, rare, and instructive.

Anatomically, "Head and Neck" clinic sessions involve the dentist most often. All patients are referred from outside sources, and from other hospital services. The emphasis is on diagnosis, treatment, and follow-up of patients with tumors of the Head and Neck region, but many non-neoplastic discrete lesions are presented for evaluation. The Departments which contribute to these sessions and the methods in which they work are these; Surgery, Dentistry, Roentgenology, Radiotherapy, Neoplastic, Radiophysics, Medicine, Ear Nose and Throat and Plastic Surgery. The patient is seen and examined by all present. X-ray series, if available, are shown and if not available are ordered. Dental films both intra and extraoral are very often employed. A brief history is given and a biopsy taken where indicated and the tentative plans of action are outlined. When pathology reports on the biopsy at the next session, the history, x-rays and pertinent data are again presented, the patient seen again and a definite course of treatment decided upon. From time to time, follow-ups on these patients are given in a report on their progress. Dental Service plays a major role in the functioning of this clinic by diagnosis, by biopsy, x-rays of mouth lesions, by referral of patients to this clinic for evaluation, by extraction of teeth in the path of radiation, by construction of stents and splints for plastic surgery procedures in the mouth, by construction of obturators and other prosthetic appliances to cover surgically created defects.

The weekly Dental Conferences are discussions of work proceeding in the dental clinic, of interesting new cases, of newer techniques in dental practice, of progress in research projects, of patients referred from other services for diagnosis, (as from "Headache Clinic" for "Temporo-Mandibular evaluation") and of any other pertinent information relative to the functioning of the dental service as an integral part of the hospital system.

It must be emphasized that the purpose of such a training program is to teach the practitioner to recognize and to diagnose and to refer to the proper physician for treatment the many cases with systemic background, that may appear before him at his office. It would serve to make the dentist aware of the precautions he must observe and of the limitations of treatment he should follow in order to fulfill his obligations as a member of the health team.

The Council on Dental Education recently sponsored a workshop on Dental Specialties. The summary of the discussions reached the conclusion that, "The hospital with teaching facilities and staff wishing to become a teaching hospital for the training of Dental Specialists should be encouraged to set up such a program."

745 Fifth Avenue
New York 22, New York

Ladies' Programme

AMERICAN ACADEMY OF DENTAL MEDICINE

12th Annual Convention

May 29, 30, 31, 1958 • Sheraton Mount Royal Hotel • Montreal, Quebec

Wednesday, May 28

12:00 Noon - 9:00 P.M. Registration...Mezzanine Floor

8-11 P.M. Hospitality Party
Salons C-D-E

Thursday, May 29

10:30 A.M. Brunch .....Salon B

1:30 P.M. Tour of new National Film Board Studios and reception

6:00 P.M. Reception .....Champlain Room

7:00 P.M. Dinner .....Champlain Room

Awards to Honorary Members Dr. Adelard Groulx and Dr. Don Gullett.

Friday, May 30

9:30 A.M. Bus tour of historic Montreal and a trip to the Montreal contribution to the St. Lawrence Seaway.

1:00 P.M. Award Luncheon
Normandie Room

All afternoon FREE for members and their spouses to explore the beauties of Montreal.

6:30 P.M. President's Reception
Champlain Room

7:30 P.M. Dinner Dance
Champlain Room

Dress optional

Saturday, May 31

9:00 A.M. - 12 Noon
Shopping in Montreal's famous Specialty Shops and Departmental Stores.

12:15 P.M. Award Luncheon
North Section of Ballroom

12TH ANNUAL CONVENTION

AMERICAN ACADEMY OF DENTAL MEDICINE

Sheraton Mount Royal Hotel Montreal, Que., Canada

May 29, 30 and 31, 1958

Dr. Albert Laferriere
3689 Park Avenue
Montreal, Canada

Dear Albert:

OF COURSE I WILL ATTEND THE ANNUAL CONVENTION!

☐ Enclosed is my Registration Fee $40.00*

☐ Enclosed is Ladies' Registration Fee $35.00*

☐ Registration Fee for Non-Member $45.00*

*This covers all fees for scientific sessions, luncheons, banquets, dances, cocktail parties, and ladies' tours.

I will bring ..... children, and will (not) need a baby sitter.

Dr. .....

Address .....

☐ I have filled out and mailed the Hotel Reservation Card.

AMERICAN ACADEM DENT

12th An nventio

WEDNESDAY, THURSDAY, FRIDAY, SATURDAY, MAY 28, 29, 30, 1958

Prime

Wednesday, May 28

12-9 P.M. Registration .....Mezzanine Floor

2:00 P.M. Board of Trustees Meeting
Salon K

8-11 P.M. Hospitality Party.....Salons C-D-E

Thursday, May 29

9:00 A.M.—
5:00 P.M. Registration
Foyer of Ballroom, 9th Floor

Scientific Session—South Section of Ballroom

TOPIC: "THE DENTIST AND THE ENDOCRINE PROBLEM."

9:00 A.M. Call to Order.....Greetings

9:30 A.M. "The Dentist Presents His Problems."

Robert F. Harvey, D.D.S. - Lecturer, Periodontia Department, Faculty of Dentistry, McGill University; Demonstrator, Department of Dentistry, Montreal General Hospital.

10:15 A.M. "THE ENDOCRINOLOGIST"

Allen Gold, M.D.C.M., M.Sc., F.R.C.P. (C). Lecturer, McGill University; Assistant Physician, Montreal General Hospital; Consultant in Endocrinology, Queen Mary Veterans' Hospital.

10:45 A.M. "THE INTERNIST"

Gerald W. Halpenny, M.D.C.M., F.R.C.P.(F.A.C.P., Hon. Sec. & Treas., Canadian Me Institute (Quebec Division), Chief, Depart of Medicine, Queen Mary Veterans' Hosp Associate Physician, Montreal General Hosp Chairman, Medical Branch, Blue Cross.

11:15 A.M. Question and Answer Period

12:15 P.M. Luncheon
North Section of Ballr

Speaker

Scientific Session—South Section of Ballr

2:30 P.M. "THE OBSTETRICIAN"

J. Lorne Macarthur, M.D., F.R.C.S.(C), F C.S., M.R.C.O.G.; Associate, Montreal Ge Hospital; Lecturer, McGill University.

3:15 P.M. "THE PHARMACOLOGIST

Kenneth I. Melville, M.Sc., M.D.C.M., Profe of Pharmacology, McGill University; Chair of Department of Pharmacology, McGill Un sity.

4:00 P.M. Question and Answer Period

4:30 P.M. "Summation"

Lester W. Burkett, A.B., D.D.S., M.D., S F.A.C.D. Dean and Professor of Oral Medi The Thomas W. Evans Museum and Denta stitute, School of Dentistry, University of P sylvania.

6:00 P.M. Reception .....Champlain R

DENTAL MEDICINE

nvention

1958 • SHERATON MOUNT ROYAL HOTEL, MONTREAL, QUE.

me

0 P.M. Dinner .....Champlain Room
Awards to Honorary Members
Dr. Adelard Groulx and Dr. Don Gullett
Business Meeting .....Brittany Room
Immediately following dinner.

Friday, May 30

0 A.M.—

0 P.M. Registration.....Foyer of Ballroom

ntific Session—Bus Transportation from el to Montreal Children's Hospital and return at noon

30 A.M. Amphitheatre of Hospital
"The Dentist and the Cleft Palate Problem."
The Cleft Palate Team of The Montreal Children's Hospital under the chairmanship of Dr. F. M. Woolhouse, Director of Plastic Surgery, the Montreal Children's Hospital.
The Team which includes some thirteen members will discuss the overall treatment plan and rehabilitation of the Cleft Palate Patient.

30 A.M. Question and Answer Period.
Return to Mount Royal Hotel by Bus.

00 P.M. Award Luncheon
Normandie Room

fternoon FREE for members and their spouses to ore the beauties of Montreal.

30 P.M. President's Reception
Champlain Room

30 P.M. Dinner Dance..Champlain Room
Dress Optional

Saturday, May 31

Scientific Session—South Section of Ballroom

"THE DENTIST AND THE DERMATOLOGY PROBLEM."

9:30 A.M. "The Inflammatory Diseases of the Mucous Membranes of the Mouth."
Lemuel P. Ereaux, M.D.C.M., Associate Professor of Medicine (Dermatology), McGill University.

10:15 A.M. "Contact Dermatitis of the Face and Hands."
Georges Leclerc, M.D., Assistant Professor of Medicine (Dermatology), University of Montreal.

11:15 A.M. Question and Answer Period.

12:15 P.M. Award Luncheon
North Section of Ballroom

2:30 P.M. Board of Trustees Meeting
Salon K

All sessions will start promptly at the times indicated.

1958 Annual Meeting Committee

LOUIS J. ROSEN ..... *General Chairman*
ALBERT LAFERRIERE ..... *Vice-Chairman, Registration, Treasurer*
ROBERT F. HARVEY, JEAN PAUL LUSSIER ..... *Scientific Programme*
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GUY LANGELIER ..... *Social Arrangements*
GEORGE F. CLARKE ..... *National Chairman*
ALFRED L. SCHERZER ..... *President, Montreal Section*

KERATOTIC LESIONS OF THE ORAL CAVITY*

DONALD A. KERR, D.D.S., M.S.†

Many lesions of the oral mucosa have the common characteristic of hyperkeratinization. An increased keratinization of the surface produces an opacity of the mucosa which results in the white appearance of these lesions. The term "leukoplakia" is frequently used to designate any lesion of the mucous membrane that is white in color regardless of the etiology or microscopic characteristics of that lesion. It literally means white plaque; however, the use of this non-specific term is of little value in making an accurate diagnosis, in treatment planning, and in considering prognosis. The term "leukoplakia" is also used to specify microscopically a hyperkeratotic lesion with cancerous potentialities, and the term is accepted at present by pathologists as a precancerous designation. The non-specific use of this term is based on a clinical impression, while the specific, precancerous designation is based on microscopic evidence. The indiscriminate label of "leukoplakia" is a dilemma in diagnosis and communication because not all white lesions are precancerous; for that reason they should not all be designated "leukoplakia." A white lesion with precancerous significance is one characterized by a disturbance of the pattern and of the metabolism of the epithelial cells; these changes of dyskeratosis can only be determined microscopically. Such terms as focal hyperkeratosis, leukoderma, and pachyderma oris should be used as clinical designations to avoid confusion in the description of white lesions of the oral mucosa until the diagnosis of leukoplakia is confirmed miscroscopically.

The white lesions of the oral cavity are manifestations of varied etiology and present a variety of clinical characteristics other than color. We will discuss the more common white lesions in which the white color is due to hyperkeratinization of the oral mucosa.

FOCAL HYPERKERATOSIS

Focal hyperkeratosis is usually associated with some type of local irritation. The irritation may be mechanical, chemical or thermal in origin. The reaction of the epithelium to mild chronic irritation is characterized by an increase in production of keratin which is retained on the surface; much like the callus produced by the dermis in response to continual mild frictional irritation. This type of lesion is frequently found on the vermilion border of the lip associated with irritation from rough or prominent teeth, certain kinds of smoking habits, or occupational activities such as "spitting" tacks or holding nails. It may occur on the gingiva or mucosa due to chronic irritation from dentures, sharp teeth, rough fillings, or may be due to chemical irritation from holding snuff or tobacco. Any source of chronic irritation may be responsible.

The lesions usually involve a small area, are sharply localized, white and slightly elevated above the surface. The mucosa is less pliable. The surface may be smooth or verrucous in character. The microscopic characteristics are variable.

One type of focal hyperkeratosis is characterized by atrophy clinically. Microscopically all layers of the epithelium are atrophic except the stratum corneum, which is increased in thickness. The epithelial cells are normal in size and regular in arrangement. The metabolism of keratin is normal except for an increased quantity of keratin on the surface due to retention. The basal layer of cells is intact, the mitotic figures are few in number or absent. There is no suggestion of proliferative activity or precancerous activity in the epithelium; however, the absence of such changes in a biopsy should not lead to a false security regarding this lesion.

* Presented before the 13th Annual Meeting of the American Institute of Dental Medicine, November 8, 1956, Palm Springs, Calif.

† Professor of Oral Pathology and Periodontia, University of Michigan School of Dentistry.

It may, due to continued irritation, develop an area of ulceration which stimulates proliferative activity and changes the character of the entire lesion as well as changing its prognosis. Focal hyperkeratosis should be treated by elimination of the chronic irritation. If the lesion does not subside following relief of chronic irritation, it should be surgically excised.

Another type of focal hyperkeratosis is characterized by epithelial proliferation. Microscopically, the epithelium is increased in thickness, the rete pegs are broad and deep with tendency for the pegs to fuse. The basal layer of the epithelium is irregular and in some cases indistinct. There are numerous but typical division figures. There is an increase in the thickness of the prickle cell and granular layers. A heavy layer of keratin of variable thickness is retained upon the surface. No dyskeratosis is evident, although the proliferative lesion may develop changes of dyskeratosis after prolonged or intense irritation. Such lesions when examined clinically have a shaggy or verrucous surface. They are somewhat elevated and firm, plaque-like in character.

The two types of lesions just described are the two prototypes of focal hyperkeratosis, and all variations between these two types may be observed. All are treated in the same manner as described for the atrophic type.

Extensive hyperkeratinization may in some cases involve the entire oral cavity and is referred to as pachyderma oris by some authors. Pachyderma oris may be associated with the excessive use of tobacco, persistent use of highly seasoned foods, irritating mouth wash or dentifrice, chronic alcoholism, and in some cases with lack of estrogenic stimulation. The microscopic changes of pachyderma oris show the many variations described for hyperkeratosis. Excision of the lesion in stages or biopsy of several areas to verify the nature of the process, followed by fulguration to complete its destruction, is indicated after all source of chronic irritation is eliminated.

LEUKOPLAKIA

This term describes white lesions of the mucosa the microscopic examination of which reveals changes in metabolism of intraepithelial hyalin. The lesions are usually characterized by an increased amount of surface keratin as well as by the presence of keratin in and between epithelial cells at all levels of the squamous mucosa. The microscopic changes of leukoplakia are difficult to describe due to the broad gamut of changes which may be observed. At one extreme there may be a mild hyperplasia with only slight increase in surface keratin. The pattern of the epithelial layers is barely disturbed, and only a limited number of cells demonstrate the inclusion of epithelial hyalin. The basal cell layer is irregular and the associated inflammation may be slight. At the other extreme there is marked alteration of epithelial pattern. There is moderate to heavy keratinization of the surface which clinically is white and shaggy or verrucous in appearance. There is marked hyperplasia of the epithelium with the production of irregular broad rete pegs which are fused. There are many division figures present. The basal layer of the epithelium is frayed and indistinct. The epithelial connective tissue boundry is obscured due to the presence of large numbers of inflammatory cells. There is almost a complete loss of orientation of the epithelial layers. The keratin metabolism is altered to the extent that masses of cells coalesce into whorls forming so-called epithelial pearls. Scattered cells filled with intraepithelial hyalin are present. In some areas the prickle cell layer is thick; in others, indistinguishable. The granular cell layer shows similar variations. The changes may be so typical of early carcinoma in some instances that they are barely indistinguishable from it. In some instances the differentiation between severe leukoplakia and early carcinoma is impossible. This blending from leukoplakia into carcinoma not only makes differential diagnosis difficult, but also presents problems in treatment planning.

A specific type of leukoplakia is associated with syphilis. The luetic glossitis that is seen in tertiary lues may be either of gummatous or sclerosing type. The entire dorsum of the tongue is involved. The dorsum becomes quite heavily keratinized, so that it is white and rough in appearance. There is some tendency

for ulceration which stimulates epithelial proliferation. In the gummatous type of luetic glossitis the tongue is nodular and often fissured. The nodules vary in size and consistency depending on the stage of the gummas. If the gumma is healed, the nodule is a firm mass of scar. If the gumma is active, the nodule is spongy or doughy and tender. In the sclerotic type the tongue is atrophic and without nodules. There is some atrophy. The dorsal surface is keratinized. There appears to be a definite association of luetic glossitis with neoplasia, and many luetics develop squamous cell carcinoma in areas of leukoplakia. These areas are very difficult to control because the entire dorsum of the tongue is involved.

PAPILLOMA

Another white lesion of the oral mucosa is the papilloma. This is a small, discrete lesion seldom more than 1 cm. in diameter. The papilloma is composed of numerous filiform or villous projections which are 1 to 3 mm. in length and 0.5 to 1 mm. in diameter arising from a common base. This lesion occurs in all areas of the oral mucosa. The initial growth of the lesion may be rapid; but after a certain size is reached, the papilloma ceases to grow and may remain in a quiescent state for several years.

The microscopic appearance of the papilloma is typical and reflects the clinical character of the lesion. It is composed of several papillomatous structures arising from a common base. The epithelial projections are long and often branching. They have a delicate connective tissue core, chiefly composed of vascular structures. They are covered by a hyperplastic squamous epithelium which shows a marked tendency to cornify. The keratohyalin is trapped between the projections of epithelium in large amounts and often constitutes a predominate part of the lesion. There are usually very few division figures seen, unless the lesion is observed in a proliferative phase, and when present, they are of normal pattern. The papilloma rarely exhibits any atypical changes or anaplastic change.

The papilloma is considered to be a benign neoplasm of squamous epithelium as the name indicates. It has been suggested that it is of viral etiology, but microscopic changes seen in the papilloma are not compatible with those seen in other virus induced lesions, especially the verruca vulgaris seen on the skin.

The sharply circumscribed superficial character of the lesion makes complete surgical excision an easy curative procedure.

SENILE OR SOLAR CHEILITIS

This is a condition frequently seen on the lips, especially the lower lip, of elderly persons. It occurs most frequently on the lips of individuals having a light skin and on those individuals who have an out-of-door occupation or avocation. There is a definite relationship between the occurence of solar cheilitis and the amount an individual is exposed to sunlight and wind. Conditioning of the mucosa by such exposure over a period of years is necessary to produce mucosal change. The intensity and duration of the exposure and degree of the pigmentation of the skin determines the age of onset of solar cheilitis. In some individuals it may be observed at 35 to 40 years of age, but it is usually seen at 55 to 60 years of age. Progress of the conditioon can be observed if the exposure is continued after its onset.

The clinical changes in the early stage are minimal. The epithelium of the lip becomes smooth and glistening. There is some tendency for tiny flakes of keratohyalin to form and separate. The lip is easily traumatized,, and frequently small, superficial ulcers or fissures appear. The vermilion border is slightly greyish and translucent. In the advanced cases the lip surface is greyish to white and the translucency is lost. There is a greater tendency for sequestration of keratin and ulceration. The lip always feels and appears dry. The patient pulls away the partially separated keratin flakes leaving superficial erosions.

The microscopic picture of solar cheilitis is quite characteristic, although it is variable in degree of severity. The epithelium shows hyperkeratosis which is often of uneven distribution. The epithelium may be reduced in thickness, and alteration in the arrangement of cell layers is present. There may be proliferation of the

basal cell layer which is often poorly defined. Near areas of ulceration, the epithelium is hyperplastic and deep rete pegs are formed which have some tendency to fuse. Hyperchromatism may be present. Some degree of dyskeratosis may also be present. The subepithelial connective tissue shows basophilic degeneration of collagen and elastic fibers. There is loss of definition and continuity of the elastic fibers and homogenization of collagen fibers. Inflammatory infiltrations, although always present, are not numerous except in association with the areas of ulceration. The combination of epithelial and connective tissue changes is quite characteristic in solar cheilitis.

There is a definite association between this form of cheilitis and carcinoma of the lip. This change is present in a large number of individuals who develop carcinoma of the lip. Bernier in a study of 766 cases of carcinoma of the lip found senile or solar cheilitis present in 83 per cent of the cases. The high incidence of association suggests that exposure and age are conditioning factors in both processes.

The treatment for solor cheilitis is protection of the lips by the use of anti-actinic creams. They serve two purposes in that they protect against the effect of sunlight and keep the lips soft. In severe cases vermilionectomy is indicated.

Because of the superimposition of carcinoma upon this change, it is well to recognize its presence early and advise the patient of the procedures which are preventive. By prevention of increased severity of solar cheilitis, the development of carcinoma may be prevented.

NICOTINE STOMATITIS

The name would indicate that this condition is produced by the action of nicotine upon the oral mucosa. This is incorrect. The changes that occur in the mouth are associated with the burning of tobacco, and both chemical and thermal factors are of significance. Nicotine stomatitis is associated with all forms of smoking, but it is most frequently associated with pipe smoking. It is most severe in those individuals who smoke rapidly and heavily. The temperature of the smoke and the concentration of combustion products are the most important considerations. However, the reaction of individuals to the combustion products of tobacco is variable. The degree of sensitivity must be considered in evaluating any specific case. The pipe smoker frequently smokes rapidly and directs the smoke through the bit of the pipe to a specific area. The smoke leaves the stem close to the tissue where the smoke is hot and the concentration of combustion products is high. In pipe smoking the combustion products are at first filtered by the tobacco in the bottom of the pipe; then as the tobacco burns towards the bottom, they are revolatilized and concentrated in the smoke. Thus, the smoke of all of the tobacco in the bowl provides excessive thermal and chemical irritation. In cigars and cigarette smoking, much of the highly volatile material is retained in the "butt;" therefore, cigar and cigarette smoking produce less irritation than pipe smoking. Evaluation of the habits of smoking are significant in making recommendations to the patient.

The changes of nicotine stomatitis are most often observed in the palate, although the gingiva and the buccal mucosa may be involved in some cases. The entire palate is usually greyish to greyish-white in appearance. The posterior one half to one third of the palate which bears accessory salivary glands exhibits red punctate dots which follow the glandular distribution. In severe cases there is a whitish nodular appearance in this area with the red dots appearing in the center of the nodule. The nodules are 1 to 2 millimeters in diameter and show a similar elevation above the surface. The buccal mucosa and gingivae show only the changes of hyperkeratosis.

The characteristic clinical appearance of nicotine stomatitis is explained by observing the microscopic features of the process. The surface of the epithelium shows increased keratinization and the epithelium is thickened; both contribute to the opacity which produces a characteristic whitish color. The ducts of the accessory salivary glands are dilated and there is metaplasia of ductal epithelium to squamous type. This produces the whitish papules with the punctate red center. There is a heavy

concentration of subepithelial inflammatory cells, predominately lymphocytes and plasma cells. The mucous glands show a chronic sialadenitis with ductal dilatation. The tissue changes are sufficiently characteristic to make a diagnosis without the aid of a case history or clinical description.

Treatment is indicated because the degree of chronic irritation exhibited in severe cases is undesirable, especially in relation to oral malignancy. Treatment is best accomplished by not smoking. However, some individuals will not stop smoking, and other procedures may be indicated. The pipe smoker can be converted to smoking cigarettes with a holder. A cigarette smoker may be converted to smoking with a holder. A holder with a filter is most effective. An acrylic or metal splint may be constructed to cover the entire palate. The splint is placed when smoking to prevent the smoke from contacting the tissue. By these methods the irritation may be reduced to a level which can be tolerated by the patient.

LICHEN PLANUS

This is a form of dermatosis which frequently has oral manifestations. The lesions of the mucous membrane may accompany the dermal lesions or appear independently. When the patient has oral manifestations without dermal lesions, he will usually seek the advice of his dentist; therefore, the disease is an oral diagnostic problem. Differentiation of lichen planus from pachyderma oris, hyperkeratosis, cheek chewing, fordyce spots, and many other conditions is necessary and sometimes difficult. It is important, however, to recognize the condition so that proper therapy may be established.

The etiology of lichen planus is unknown, but it occurs most frequently in the third or fourth decade of life with the incidence being somewhat higher in females. The patients usually have an emotional temperament, are under nervous strain, or have numerous anxieties. Emotional strain and anxieties produce exacerbations of the disease. The patients usually have a well developed cancerphobia that in many instances has been intensified by a faulty diagnosis of leukoplakia. Burket states that 50 per cent of the patients having skin lesions have oral manifestations, but that less than 10 per cent who present with oral lesions have dermal lesions. Many patients having lesions of the oral mucosa also have other mucosal surfaces involved.

The oral lesions of lichen planus involve the buccal mucosa most often; but the tongue, lips, gingiva, palate, and floor of the mouth may exhibit the condition. The lesions of the buccal mucosa are the most typical. They are usually asymptomatic, but occasionally they are pruritic. They are usually called to the patient's attention by roughness of the mucosa or by chance observation. The characteristic lesion of the buccal mucosa appears in the molar and retromolar area as slightly elevated, greyish or bluish-white fine lines (Wickham's striae). They exhibit a pattern of arborization with a small elevated spot 0.5 to 1.0 millimeters in diameter at the area where the lines cross or meet. In some instances the lines form a stellate pattern. In very severe cases the lesions may be plaque-like geometric figures at the border of which the characteristic striae can be observed. The lesions of the gingivae and tongue are most often plaque-like in type and have a linear or rhomboid shape. They vary in size from a few millimeters across to involvement of the entire gingiva or dorsum of the tongue. Unlike pachyderma oris and hyperkeratosis, the mucosae in lichen planus is of normal elasticity. In some cases the surface of the lesion is macerated producing an erosive type of lesion. This is most frequently observed in the buccal mucosae and gingivae. The erosive gingival lesions are frequently mistaken for desquamative gingivitis. The erosive phase of the disease is probably due to the moist nature of the environment and the effect of trauma. The erosive lesions are tender, and there is burning and smarting, especially on taking food. The erosive lesions show exacerbations and remissions as do the non-erosive type.

The microscopic changes in non-erosive lichen planus are quite characteristic, and biopsy to demonstrate these findings is therefore helpful in establishing the diagnosis. In the erosive type of the superimposed inflammatory change

complicates the picture and makes microscopic diagnosis unsatisfactory unless tissue is removed some distance from the erosion. The changes observed are those of hyperkeratosis and parakeratosis, which in the lace-like lesions are focal in character. There is inter- and intracellular edema of the prickle cell layer and fraying of the basal cell layer. Inflammatory infiltrations, chiefly lymphocytes, are numerous in the submucosa. They are sharply limited to the epithelial connective tissue junction and extend over into the mucosa. This accentuates the indistinctness of the basal cell layer. There is submucosal edema and slight dilatation of capillaries. These changes may be confused with hyperkeratosis having an associated inflammation. However, the spongiosis and location of the inflammatory cells present in lichen planus are helpful in differentiation from focal hyperkeratosis.

The treatment of lichen planus is in general unsatisfactory, especially in those cases having only mucous membrane lesions and where the oral lesions appear before the skin lesions. The mucous membrane lesions do not respond to therapy as readily as do the skin lesions. The treatment should be directed toward the patient rather than the disease. The use of tranquilizing drugs and barbiturates to reduce emotional anxiety, the assurance that the disease is benign, and in some cases psychotherapy is indicated. The use of bismuth is helpful, but it is more effective where the skin is involved. The recognition that exacerbations and remissions reflect the emotional status of the patient is important, and this relationship should be pointed out to him. Observation of the lesions at three to six month intervals is desirable to alleviate cancerphobia.

The treatment of the erosive type of lichen planus is even more unsatisfactory than the nonerosive type because of the superimposed infection. Pain can be controlled by the use of syrup of phenergan or viscous Xylocaine. Elimination of trauma and local irritation is necessary. This type of the disease usually does not respond to bismuth or other forms of medication.

LUPUS ERYTHEMATOSIS

This is a form of dermatosis which may be either chronic (discoid type) or acute (disseminate type). The process may be present as a fixed eruption for a period of years and then change to the disseminate type with involvement of the viscera. The disseminate type runs an acute course and is usually fatal. The disease is of unknown etiology, but belongs in the group of collagen diseases. There is a strong suggestion that the disease is allergic in character. In some instances there appears to be a hypersensitivity to sunlight indicated by exacerbations which follow exposure to the sun. The discoid involves the skin of the body, especially the face. The lesions are usually sharply demarcated pinkish or bluish patches which are slightly elevated.

Oral manifestations occur in a small percentage of the cases. According to Bernier, 50 per cent of patients have oral manifestations; Burket places the oral incidence at 15 per cent, while Allen gives a more realistic figure, indicating that 25 per cent demonstrate co-existence of cutaneous and oral lesions. All areas of the oral mucosa may be involved, but gingival involvement is rare. The skin lesions usually appear before the oral lesions, and their presence is most helpful in establishing the diagnosis. The oral lesions are well defined, slightly elevated hyperkeratotic patches with dark reddish-purple margins often accentuated with radiating telangiectasis which may produce bleeding points. The older lesions have a tendency to become ulcerated and heal with mild scarring.

Biopsy is an aid in the diagnosis of lupus erythematosis, although the mucosal changes are not as typical as the skin changes. Alternating areas of acanthosis and atrophy with an associated hyperkeratosis, liquefaction of the basal cell layer, hyperemia of the papillary layer of the mucosa are seen, as well as dense collections of mononuclear cells, especially lymphocytes, in the upper and mid portions of the mucosa. In the skin keratotic plugging of the hair follicles and the tendency for inflammatory cells to concentrate about the appendage are valuable aids in diagnosis.

The treatment of lupus erythematosis is unsuccessful, but the best palliative results are obtained by the use of steroids.

HAIRY TONGUE

This condition is produced by hyperplasia of the filiform papillae which becomes elongated and appear as black or dark brown hair-like structures. It may develop in a short period of time and disappear spontaneously after having been present for a period of years. It is believed to be due to irritation caused by chromogenic organisms and fungi or by various drugs.

Various manifestations of hairy tongue may be produced by a variety of causes. It is in some instances idiopathic, but the condition accompanies various forms of antibiotic therapy, the prolonged use of 3 per cent hydrogen peroxide as a mouth wash, and the presence of various forms of fungi, especially monilia-type organisms. The presence of these organisms seem to be an important factor. They are not affected by the antibiotics, but increase in number as the other oral flora is diminished. The condition usually persists for a short time after antibiotic therapy is terminated, but disappears as the oral flora returns to normal.

The clinical picture is one of black hair-like structures projecting from the dorsum of the tongue. The process is often limited to areas where there are deep folds, or the entire tongue may be involved. The process is more intense in the areas of fissures. The "hairs" in most cases are 2 to 4 millimeters in length, but in unusual cases they may be a centimeter in length. There are rarely any associated symptoms except when the hairs are very long and touch the palate. When the hairs desquamate following antibiotic therapy, there may be a rather severe transient glossodynia.

Treatment depends on the etiology. Elimination of antibiotics or peroxide, reduction in sugar intake, the use of fungicides (sodium caprilate) and occasional use of salicylic acid are all employed.

WHITE SPONGY NEVUS

This name was suggested by Cannon in his case reports of 1935. Orban designated the lesion as white folded gingivostomatitis.

White spongy nevus is a rare condition of the oral and other mucosal surfaces which is characterized by changes of a hyperkeratotic nature which involve the buccal mucosa, tongue, floor of the mouth, and gingiva. The condition is asymptomatic, and the patient may be aware of its presence only because of the feeling of dryness or roughness of the mucosa. It is usually discovered on routine examination and called to the attention of the patient. The examiner is no doubt deeply concerned because of the extensive involvement and the character of the change. The involved mucosa appears intensely white or opalescent in color. The surface is shaggy or sponge-like in character. The mucosa appears thickened. The consistency of the tissue appears normal. At first it may suggest one of the various forms of hyperkeratosis, but close inspection shows the typical porous or spongy character of the lesion.

The histologic changes are limited to the epithelium which is markedly thickened. The rete pegs are broad and fused. The basal layer is usually well defined and intact. There is acanthosis and marked spongiosis. The prickle cell layer which is increased in width shows marked edema and hydropic degeneration. There may be slight dyskeratosis. Some of the swollen cells contain fine granules. The granular layer is absent, and the surface is composed of a thick parakeratotic layer. At intervals throughout the epithelium there are depressions filled with parakeratotic plugs. The parakeratotic surface is irregular in contour.

White spongy nevus is familial in character, and several members of succesive generations of a family exhibit varying degrees of change. The familial history is of great significance in diagnosis. Because the condition simulates leukoplakia, the observer becomes alarmed regarding the extensive nature of the disease and the precancerous potentialities carried by the diagnosis of leukoplakia. Biopsy reveals the true character of the disease. From the available evidence, carcinoma is not associated with this condition. There is no indicated treatment, but it is desirable to observe the patient at intervals.

SUMMARY

As is evident from this discussion, there are numerous so-called keratotic lesions all of which exhibit the common characteristic of hyperkeratinization. The whitish appearance due to the hyperkeratinization produces a clinical similarity which makes differential diagnosis difficult, especially in distinguishing between the lesions that show dyskeratosis and those that do not. It also makes difficult the distinction between lesions which are of local significance and those which represent oral manifestations of a systemic disease.

The differential diagnosis of these lesions is of great importance in outlining a treatment plan and determining the prognosis for the patient. If the lesion has a precancerous significance, it is imperative that it be eliminated rather than allowed to progress and develop malignant changes. It is also important to recognize those conditions which represent only one aspect of a systemic disease so that the disease may be treated rather than the individual lesion. Because dyskeratosis cannot be determined by clinical observation, biopsy is a necessary diagnostic procedure to prove or disprove the precancerous nature of a specific lesion. Biopsy is a procedure which also may be of value in determining the systemic nature of a lesion. Whenever the clinical characteristics of a lesion do not permit a definitive diagnosis, biopsy should be performed. Definitive diagnosis of these keratotic lesions is necessary if we are to be the physicians of the oral cavity and provide for our patients the health service which their confidence in us demands.

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Author's Note: This is not a comprehensive discussion of all hyperkeratotic lesions found in the oral cavity, but rather a discussion of the more common ones which could be discussed in the time allotted during the seminar.)

LESIONES KERATOTIC DEL CAVITATE ORAL

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SUMMARIO IN INTERLINGUA

Multe lesiones del mucosa oral ha in commun le characteristica de hyperkeratinisation. Augmentate keratinisation del superficie produce un opacitate del mucosa que es responsabile pro le apparentia blanc de iste lesiones. Le termino "leucoplasia" es frequentemente usate pro designar omne lesion del membrana mucose que exhibi un color blanc, sin reguardo al etiologia o al characteristicas microscopic del lesion in question. Le termino "leucoplasia" es etiam usate pro specificar que un lesion hyperkeratotic exhibi sub le microscopio signos de potentialitates cancerose. Pathologos usa le termino currentemente pro designar un condition pre-cancerose. Le uso non-specific del termino reflecte un impression clinic; su uso specific—como designation de un stato pre-cancerose—se basa super evidentia microscopic. Le promiscue uso del etiquette "leucoplasia" constitue un dilemma diagnostic e communicatori, proque non omne lesiones de color blanc es pre-cancerose. Ergo il esserea desirabile non designar omnes como "leucoplasia." Un lesion blanc con signification pre-cancerose es un lesion characterisate per un disturbation del configuration e del metabolismo del cellulas epithelial. Tal alterationes dyskeratotic es

determinabile solmente per medio de technicas microscopic. Terminos del typo de hyperkeratosis focal, leucoderma, e pachyderma oral deberea esser usate como designationes clinic pro evitar omne confusion in le description de lesiones blanc del mucosa oral usque le investigation microscopic confirma le diagnose de ver leucoplasia (con potentialitates cancerose).

Le diagnose differential del lesiones hic discutite es de grande importantia como base del programma therapeutic e del prognose in le caso individual. Si le lesion representa le typo a signification pre-cancerose, il es un desiderato urgente excider lo e non permitter a illo progreder e disveloppar alterationes maligne. Il es etiam importante recognoscer le conditiones que representa solmente un aspecto de un disordine systemic, proque alora le morbo systemic debe esser tractate plus tosto que le lesion individual. Proque dyskeratosis non pote esser determinate per observationes clinic, biopsia es un mesura indispensabile pro establir le diagnose positive o negative del natura pre-cancerose de un lesion particular. Biopsia es etiam un mesura que pote esser de valor in determinar le origine systemic de un lesion. In omne caso in que le characteristicas clinic de un lesion non permitte un diagnose definitive, le effectuation de un biopsia es urgentemente recommendabile. Le diagnose definitive de iste lesiones keratotic es necessari si nos vole esser le medicos del cavitate oral e si nos vole offerer a nostre patientes un servicio de sanitate que merita lor confidentia.

TISSUE REACTION TO DENTAL CALCULUS AND TO FILLING MATERIALS*

HELMUT A. ZANDER, D.D.S., M.S.†

If one assumes the healthy gingivae of a young adult to be "normal", then several clinical morphological and color factors are evident. The gingival tissues hug the teeth tightly in a cuff-like fashion. They terminate in the interproximal spaces in a knife-like edge. They are coral pink and firm. A sharp line of demarcation exists between attached gingival tissues and the alveolar mucosa. The color of the alveolar mucosa is somewhat darker, toward the red, than the attached gingival tissues. Clinical reaction to dental calculus manifests itself by changes of the gingivae in color and form. Progressive darkening of the gingivae, from the normal pink toward red or blue, and either form change of enlargement or recession of tissue occur. An early sign of gingival involvement is loss of the knife-like edge and a distinct roundness of the gingival margin. These clinical manifestations are based upon the underlying histological changes which can be summed up under the heading of "tissue reaction to dental calculus and to filling materials."

CHANGES IN PAPILLA

A section from the interdental papilla reveals that the place once occupied by the knife-edged papillary gingiva is now filled with calculus. (Fig. 1.) The gingival papilla has changed in contour and follows in outline the lower border of the calculus. On one side the epithelial attachment is intact on the enamel and terminates about at the cemento-enamel junction. On the adjacent tooth, calculus has already reached the cemento-enamel junction and the epithelium has migrated along the cementum of the root surface of this tooth. Normally the connective tissue of the marginal gingiva is covered by a thick layer of stratified squamous epithelium. This epithelium is well differentiated into its various layers—the basal, prickle-cell, granulated, and keratinized layers. The connective tissue usually contains dense collagenous fibers. The tissue reaction to calculus has resulted in a loss of the well-defined layers of the epithelial covering. One no longer can distinguish the different layers. The epithelium is very thin and may be entirely lacking in some areas presenting an ulcerated surface. Many fine extensions of the epithelium, called "rete pegs", can be seen deep in the connective tissue corium. It is permeated by many inflammatory cells. These cells are not only in the epithelium, but accumulations of these cells are also found between the epithelium and the calculus as well as in the underlying connective tissue. The type of cells predominating may be polymorphonuclear leukocytes, lymphocytes, macrophages, or plasma cells, depending upon the acuteness or chronicity of the tissue reaction.

Concomitant with the appearance of these cells the collagenous fibers tend to disappear and a vascular reaction of dilation and increased permeability takes place. Also, many new capillaries are found close to the tissue surface.

These described tissue reactions are typical of inflammation. Of interest to us is the fact that as long as calculus is present the vascular reaction and exudation in the gingivae do not result in resolution and repair as ordinarily occurs in inflammatory processes. The understanding of the tissue reaction to these exciting factors makes this behavior of the gingivae understandable.

CALCULUS IS EXCITING FACTOR

The exciting factor is calculus. Calculus consists of an organic matrix which is made up primarily of micro-organisms. The predominant

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Fig. 1a. Interdental area between two incisors.
C—Calculus, E—Enamel.

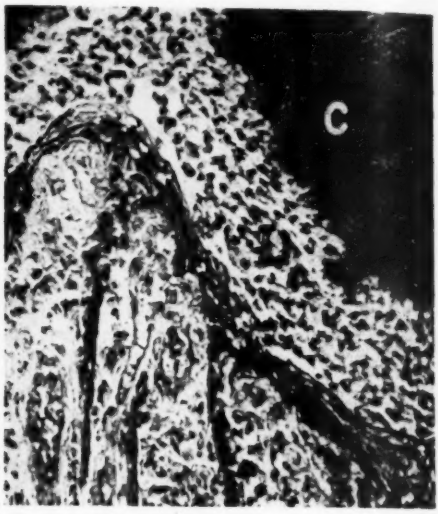

Fig. 1b. Higher magnification (from center of Fig. 1a) of area between the calculus and the gingiva.
C—Calculus, I—Intermediary zone—area between calculus and gingiva.

organisms are of filamentous character. They have been classified in the genera leptotrichea or actinomyces. These organisms possess the ability to attach themselves to smooth surfaces and to precipitate substances which can calcify. It is not yet entirely clear whether or not these organisms cause only calculus attachment, paving the way for symbiotic association with other micro-organisms on the tooth surface, or if they themselves also contribute, through their enzyme systems, to the tissue reaction of the gingivae.

The work by Schultz-Haudt[1-4] has been a help toward elucidating the role that the micro-organisms in calculus and plaque material may play in eliciting the tissue reaction. It has been shown that many of the organisms can produce hyaluronidase, beta-glucoronidase, phenolsulfatase, and chondrosulfatase. These enzymes are capable of catalyzing the breakdown of mucopolysaccharides which are tissue constituents. In trying to understand the tissue reaction to dental calculus, we must discontinue the concept of a mechanical irritation eliciting the specific inflammatory response. Instead, we must look upon the tissue defense of inflammation as a response to bacterial-chemical agents. Ordinarily the tissue cells—leukocytes and lymphocytes—cope with this type of attack by producing factors that destroy the enzymes liberated by the micro-organisms. Even when these cells have lost their contact with the tissue from which they originate, their breakdown products are shown to have similar properties. In addition, macrophages are capable of engulfing micro-organisms once they have penetrated the tissue. However, when calculus causes the reaction, both mechanisms of inflammatory defense are unsuccessful because of the peculiar way in which calculus is attached to the tooth surface. In simple words, they can never "get at" these micro-organisms because they have their backs against the inert tissue of enamel or cementum, which does not have the ability to counteract them. This point can best be illus-

trated by a study of the mode of calculus attachment to tooth surfaces.[5]

TYPES OF CALCULUS ATTACHMENT

We recognize four main types or combinations of calculus attachment. One, calculus may lie on the dental cuticle. Two, it may have developed on an irregular cementum surface. Three, the organisms may actually have penetrated into the cementum. And, four, they may have grown into various cementum resorption areas that developed prior to the exposure of the root surface to conditions favorable to the formation of calculus. Looking at histological sections of calculus attachment, one can easily understand why the defense mechanisms of gingival inflammation are limited to the first two stages of vascular reaction and exudation and never result in resolution and repair unless the dentist removes the exciting cause; namely, the calculus.

Some clinical considerations for the removal of calculus are obvious when one better understands the role of tissue reaction and the modes of calculus attachment. One, it may be relatively easy to remove calculus from the tooth surface when we deal with modes "one" and "two" of calculus attachment. Two, it may be impossible to remove all of the calculus without injuring and removing part of the tooth surface —in particular, the cementum. This has been shown by a study conducted by E. M. Schaffer[6] in which teeth were curetted in the mouth before removal. Afterwards these teeth were studied under the dissecting microscope and sectioned histologically. It was found that only diligent therapeutic planing of the root surface resulted in the removal of all calculus. In almost every one of these cases all of the cementum was also removed in the process. The significance of this finding will be discussed under the heading of "mechanism of healing" in another lecture.

CLINICAL FINDINGS OF GINGIVAL INFLAMMATION

The clinical finding of gingival inflammation around restorations is familiar to every practicing dentist. It is classified as a mechanical irritant and, in order to prevent gingival irritation, we try not to have "overhanging margins." A study of filling materials which were placed beneath the gingival margin of monkey teeth, surprisingly enough, did not show as much of an injury due to the overhanging margin of restorations as to the material itself or the microscopic plaque which accumulated on the surface of the filling material.[7,8] The irritating qualities of silicate cement have been well documented in regard to its effects upon the dental pulp. Gingival tissues which come in contact with this material present the picture of a chronic inflammation characterized by an abundance of lymphocytes and changes in the protective layer of epithelium. This does not hold true for acrylics, porcelain, and gold where the epithelial layer injured during cavity preparation heals readily, forming a new attachment to the filling material in much the same way as to tooth surface. If, however, micro-organisms attach themselves to the surfaces of such restorations before the epithelium has had a chance to regenerate, very much the same type of tissue reaction occurs as to calculus. Amalgam may, by itself, elicit a chronic inflamatory reaction which is not as severe as the one seen to silicate cements. The most common finding in the histological sections was the presence of a crevice between the given filling material and the cavity wall. Some of these crevices were as small as 0.02 mm but ranged to 0.36 mm in a few instances. The fact that small crevices exist between restoration and tooth has been well documented by studies at the Bureau of Standards[9] where it was called "marginal percolation" and by studies at Minnesota[10,11] on penetration of radiocalcium between tooth and restoration. However, we know now that these microscopic crevices located below the gingival margin are filled with mirco-organisms of the same type found in the calculus plaques. This is the most significant factor responsible for the similarity of the reaction of periodontal tissue to calculus and to dental restorations.

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HISTOREACTIONES A CALCULO DENTAL E A MATERIALES DE PLOMBAGE

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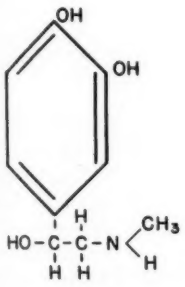
SUMMARIO IN INTERLINGUA

Un section del papilla interdental (Fig. 1) revela que le placia olim occupate per le cresta del gingiva papillari es nunc plenate de calculo. Le papilla gingival ha alterate su contornos e seque le linea del margine inferior del calculo. A un latere, le attachamento epithelial es intacte super le email e se termina plus o minus al junction inter cemento e email. In le caso del dente adjacente, le calculo ha jam attingite le junction inter cemento e email, e le epithelio ha migrate sur le cemento del superficie de radice de iste dente. Normalmente le histos conjunctive del gingiva marginal es coperite per un spisse revestimento de stratificate epithelio squamose. Iste epithelio es ben differentiate in su varie stratos de cellulas basal, spinose, granulate, e ceratinisate. Le histos conjunctive contine usualmente dense fibras collagenic. Le historeaction al calculo ha resultate in un perdita del ben-definite stratos de revestimento epithelial. Il ha devenite impossibile distinguer le differente stratos. Le epithelio es multo tenue; in certe areas con superficies ulcerate, illo pote esser completemente absente. Numerose extensiones delicate del epithelio pote esser vidite profunde in le corio del histos conjunctive. Illo es permeate de multe cellulas inflammatori. Iste cellulas non se trova exclusivemente in le epithelio, sed accumulationes de illos es incontrate inter le epithelio e le calculo e etiam in le subjacente histos conjunctive. Le typo predominante de cellula pote esser leucocytos polymorphonucleari, lymphocytos, macrophagos, o plasmocytos, dependente del acutessa o chronicitate del historeaction.

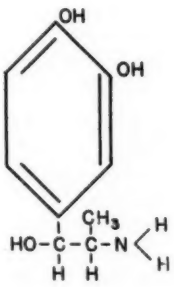
A CLINICAL REPORT OF L-NORDEFRIN AS A VASOCONSTRICTOR IN LOCAL ANESTHESIA

EDWARD C. DOBBS, B.S., D.D.S.*, NORTON M. ROSS, B.S., D.D.S.**, and STANLEY L. BROWN, B.S., D.D.S.†

Levo-nordefrin hydrochloride (L-Cobefrin, Neo-Cobefrin) was synthesized at the Sterling-Winthrop Research Institute in 1951. Chemically it is the levo-rotatory isomer of l- (3, 4 -dihydroxyphenyl) 2 amino-propanol hydrochloride with an optical rotation of -29-31°. It is a chemical isomer of epinephrine and mimics its physiologic actions. Physically it occurs as an almost microcrystalline solid with a melting point of 208-215°C (Corr.) The base is almost insoluble in distilled water, but the hydrochloride salt is freely soluble.[1]



EPINEPHRINE



NORDEFRIN

Pharmacodynamically, the site of action appears to be on the effector cells at the sympathetic myo- and adeno- neural junctions. Its mode of action is sympathomimetic acting directly, being effective on tissues which have been denervated.

While l-nordefrin is a comparably new compound, its racemic mixture (d-l-nordefrin, Cobefrin) has been used as a vasoconstrictor in local anesthesia since 1935, and, in a few instances, is used for comparative purposes in this paper. Racemic nordefrin contains about equal volumes of the d- and l- isomers, and as most of the biological activity resides in the l-form, it is only one half as active and toxic. Schaumann,[2] in his blood pressure studies on anesthetized cats using r- and l- nordefrin, found the ratio of pressure activity to be 1:1.6; or a 1:10,000 solution of the racemic to be equivalent in pressor principle to a 1:16,000 solution of the l-form.

Comparative studies of the l- and d- isomers of nordefrin by Schaumann[3] demonstrated the l- form to have 160 to 200 times the pressor effect. He also found that after cocainization the activity of the l-form was potentiated, while the d- form was not. This result suggests a different pathway of action for the two isomers. The blood sugar was increased 40 per cent with the l- form and only 20 per cent with the d- form. Table 1 is a partial report on the subcutaneous (s.c.) and the intravenous (i.v.) lethal doses in mice:

TABLE 1

Drugs	*Route*	*mg/kg*
l-form	i.v.	8
l-form	s.c.	70
d-form	i.v.	200
d-form	s.c.	800

These figures give the i.v. and s.c. lethal doses of l- and d- cobefrin as 1 to 25 (i.v.) and 1 to 11 (s.c.).

Luduena *et al*,[4] in their studies of the optical isomers of nordefrin in pentobarbital anesthetized dogs, reported that the l-form produced a rapid rise in blood pressure over the pre-injection normal within 2 to 2.5 minutes, was of short duration and was sometimes followed by a slight fall in pressure. There was cardiac acceleration also. The latter results are misleading

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physiologically and clinically, as the parasympathetic nervous system influence to the heart was blocked with a parasympatholytic drug [2 diethyl-aminoethyl - 2 - cyclopentyl (2-thienyl) hydroxy acetate methobromide] in this experiment.

The d-form under the same experimental conditions with comparable blood pressure dosages gave a longer onset and duration time. These results, again suggest that the mode of action of the two isomers may be different.

Table 2, taken from the above quoted data, shows the i.v. toxicity in mice and that the l-isomer is 10 times as toxic as the d-form.

TABLE 2

Drug	*No. Animals*	*L.D. 50 + s.e. mcg./kg. of base 24 hrs.*
d-nordefrin	50	128 ± 13
l-nordefrin	40	12.6 ± 2.7

A comparative study of the vasoconstrictor efficacy of two local anesthetic preparations (propoxycaine HCl 0.4% and procaine HCl 2.0%) when the only variables were d-l-nordefrin or l-nordefrin has been reported.[5] The experimental animals were guinea pigs; the method used was the intradermal wheal test, and the presence of anesthesia was determined by pinpricking. They found that the anesthetic potency of the l-form in a 1:20,000 solution was similar to that of the d-l-form in a 1:10,000 solution.

A bio-assay stability test on levo-nordefrin solutions containing local anesthetics (see above) showed no loss of pressor activity in 2 years at room temperatures, indicating no measurable racemization at that time-temperature interval.

With these facts in mind we began our laboratory and clinical screening of l-nordefrin in a 1:20,000 solution combined with propoxycaine HCl (Ravocaine) 0.4 per cent and procaine HCl (Novocain) 2.0 per cent and compared the results with a known proprietary solution in which the only variable was the vasoconstrictor (d-l-nordefrin HCl 1:10,000)[6]

Table 3 shows a comparative intraperitoneal toxicity study which was done on 25 Gm. mice using three dosage forms for each solution:

TABLE 3

Sol. Used	*Amount Injected*	*No. of Mice*	*Dead in 15 min*	*% survived 15 min*
1	0.5 ml.	8	8	0
1	0.3 ml.	9	7	22
1	0.25 ml.	18	10	44
2	0.5 ml.	8	8	0
2	0.3 ml.	9	9	0
2	0.25 ml.	18	15	17

Solution 1 is with 1-nordefrin 0.005%, propoxycaine HCl 0.4% and procaine HCl 2.0%. Solution 2 is with d-1 nordefrin 0.01%, propoxycaine HCl 0.4% and procaine HCl 2.0%.

These results suggest that when equipressor concentrations of the l-form is used as the vasoconstrictor and compared with the d-l-form the i.p. toxicity in mice is lower.

The circulatory effects of solution #1 (See Table 3) were tested on 8 dental students by infiltration injections of 2.2 ml. of the prepared solution into the mucolabial fold in the inferior maxillary incisor area. In an attempt to minimize the apprehension, no clinical work was done on these students. Two readings each of blood pressures (systolic and diastolic) and of pulse rate were taken at 2-minute intervals before injection (normal), and for 5 two minute intervals following injection. The blood pressure readings were taken with a Tycos clock manometer, the algebraic average (sum of + and —, ÷ by the no. of readings) being —2 mm. (extremes — 8 to + 6 mm. of Hg.) for the systolic and + 1 mm. (extremes — 4 to + 5 mm. of Hg.) for the diastolic. The averaged pulse changes also varied very slightly from the normal, the algebraic average being +2.5 beats per minute (extremes —4 to +5 beats). There was a tendency for a slight rise in systolic and diastolic blood pressures and pulse rates for the first 4 minutes followed by a slight fall in pressures and rates during the next 6 minutes.

To compare the anesthetic properties of solutions 1 and 2, (see Table 3), 100 clinic patients (operative dentistry) were used. The prepared solutions[7] were injected by senior dental students. It is conceded that the injection technics were not perfect; however, the results are comparable for such a screening procedure. The data on solution 2 were collected about 14 months earlier during a similar experiment (see Table 4).

TABLE 4

	Sol. 1	*Sol. 2*
Satisfactory anes.	80%	79%
Pt. felt some pain	18%	7%
Pt. reinjected	2%	14%
Average onset time	68 sec.	63 sec.
Average duration of anes.	59 min.	60 min.
Average duration of numb.	149 min.	157 min.
Average amt. injected	1.8 ml.	2.09 ml.

There were no complaints of pain at the time or following the injections with either solution, a result which confirms the trypan blue local irritation studies on albino rabbits of Hoppe *et al.*[8]

CONCLUSIONS

It appears from our screening procedure that levo-nordefrin is a satisfactory vasoconstrictor for use in prepared local anesthetic preparations in a concentration of 0.005% or 1:20,000 concentration when combined with propoxycaine HCl 0.4% and procaine HCl 2.0%. As the d-form has practically no vasoconstrictor properties, its use in a prepared local anesthetic mixture can only exaggerate the undesirable reactions and, therefore, is better omitted.

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REPORTO CLINIC IN RE L-NORDEFRINA COMO VASOCONSTRICTOR IN ANESTHESIA LOCAL

Dr. chir. dent. Edward C. Dobbs (B.S.), Dr. chir. dent. Norton M. Ross (B.S.), e Dr. chir. dent. Stanley L. Brown (B.S.)

SUMMARIO IN INTERLINGUA

Hydrochloruro de levo-nordefrina (L-Cobefrina, Neo-Cobefrina) esseva synthetisate in 1951 al Instituto de Recercas Sterling-Winthrop. Chimicamente illo es le isomero levo-rotatori de hydrochloruro de 1-(3,4-dihydroxyphenyl)-2-amino-propanol, con un rotation optic de -29-31°. Illo es un isomero chimic de epinephrina e simula le actiones physiologic de illo. Physicamente illo se presenta como un solido quasi microcrystallin con un puncto de fusion de 208-215 C. Le base es quasi insolubile in aqua distillate, sed le sal hydrochloruric es liberemente solubile.

Ab le puncto de vista pharmacodynamic, le sito del action es apparentemente le cellulas effectori al junctiones sympathic myo- e adeno-neural. Su modo de action es sympathomimetic e affice directemente le histos que ha essite disnervate.

Il resulta ab nostre essayos que levo-nordefrina es un agente satisfactori de vasoconstriction pro le uso in prefabricate preparatos de anesthesia local in un concentration de 0,005 pro cento

(1:20.000) quando combinate con hydrochloruro de propoxycaina (0,4%) e hydrochloruro de procaina (2,0%). Viste que le forma dextero-rotatori ha practicamente nulle qualitates vasoconstrictori, su uso in le preparation de mixturas de anesthesia local pote solmente exaggerar le reactiones adverse. Ergo il vale melio omitter lo.

SYMPOSIUM ON TEMPOROMANDIBULAR JOINT DISORDERS

Friday, May 2, 1958

sponsored by

Veterans Administration Hospital, Dental Service
Brooklyn, N. Y.
and
The Second District Dental Society of New York

Sessions will be held at the Veterans Administration Hospital, 800 Poly Place, Brooklyn 9, N. Y.

This is the 7th annual symposium devoted to a specific medico-dental problem. Its purpose is to review present-day knowledge on the subject from various points of view and to consider its application to modern dental practices.

The subjects selected for these symposia are often controversial and it would be impossible to touch upon all of their scientific and clinical aspects in one day. Accordingly, the morning session will review some of the basic science and theoretical considerations and the afternoon session will be devoted to the rationale of modern clinical practices. Each of the participants is outstanding in his field and has made significant contributions toward understanding of the problems of temporomandibular function and disorders.

Morning Program 9:30-12:30

Chairman: LESTER R. CAHN, D.D.S., F.D.S., R.C.S. (Eng.)
Member of Deans Committee and Consultant in Oral Pathology, V.A. Hospital, Brooklyn, N. Y.

Introduction: ALEXANDER W. KRUGER, M.D.
Manager, V.A. Hospital, Brooklyn, N. Y.

Anatomy of the Temporomandibular Joint and Associated Structures:
HARRY H. SHAPIRO, D.M.D.
Associate Professor of Anatomy, College of Physician and Surgeons, Columbia University.

Neuromuscular Mechanisms Involved in Mandibular Movements:
NICHOLAS A. DI SALVO, D.D.S., Ph.D.
Associate Professor of Dentistry; Director, Division of Orthodontics, Columbia University.

Pathology of the Temporomandibular Joint:
LASZLO SCHWARTZ, D.D.S.
Clinical Professor of Dentistry; Director, Division of Clinical Oral Physiology, Columbia University.

Afternoon Program 1:30-4:30

Chairman: J. BARDIN GOODMAN, D.D.S.
President, Second District Dental Society.

Each speaker will present a brief summary of the clinical problems of temporomandibular disorders as viewed from his specialty, and a panel discussion will follow.

Morphological and Anatomical Changes After Occlusal Reorientation:
SIDNEY E. RIESNER, D.D.S., F.A.C.D
Lecturer, Schools of Dentistry, Tufts University and University of Pennsylvania.

The Treatment of Temporomandibular Pain Syndrome:
HARRY M. SELDIN, D.D.S., F.A.C.D., F.I.C.D.
Diplomate, American Board of Oral Surgery.

General Practitioner Views Problems of Temporomandibular Disorders:
IRVING SHEPPARD, D.M.D.
Former Research Fellow, Montefiore Hospital, Bronx, New York.

The Role of Occlusal Rehabilitation in Temporomandibular Disorders:
ERNEST R. GRANGER, D.D.S., F.A.C.D.
Associate Professor, School of Dentistry, University of Pennsylvania.

Case Report

BONE REGENERATION WITH CONSERVATIVE PERIODONTAL TREATMENT

Abraham Goldstein, B.S., D.D.S., F.A.D.M.*

HISTORY AND DIAGNOSIS

A patient, male, age 38, presented for treatment for pain and swelling on the lingual aspect of the gingiva in the region of the mandibular left lateral incisor and canine.

Clinical examination revealed an acute swelling, severe pain and class 3 mobility of the left lateral incisor.

History revealed no symptoms until the previous day, when a slight "awareness" of the incisor had been felt. The patient had not been conscious of any mobility. Subsequent roentgenographic examination showed a normal bone level and texture throughout the mouth with the exception of the left incisor-canine region (Figure 1).

Further clinical examination and study of casts revealed that the entire occlusion needed correction by means of equilibration and, specifically, that the left canine and lateral incisor were in trauma in protrusive excursion and position and also in left lateral excursion.

Calculary deposits were present throughout the mouth. Pockets which were present were of the horizontal, shallow type with the exception of the left lateral incisor-canine region. A deep pocket was present between these teeth. It was still present after the periodontal abscess subsided.

The main etiologic factor was traumatic occlusion.

TREATMENT

The periodontal abscess was opened at the gingival margin revealing a purulent exudate.

With a number 11 diamond stone, the incisal edges of the left lateral incisor and canine teeth were reduced until contact was divided over many more teeth. The lingual inclined planes of the maxillary incisors and canines were ground to permit freedom of movement in the protrusive and lateral excursions.

The patient was dismissed with instructions to use hot hypertonic saline irrigations every hour and to return the next day.

At the second visit, the acute symptoms had subsided and a full series of roentgenograms and impressions for study casts were taken. The area of the periodontal abscess was curetted carefully and a wire splint was placed on the mandibular teeth extending from left second premolar to right canine.

Routine conservative periodontal procedures were then instituted, consisting of scaling, toothbrushing instruction, dietary analysis and correction, further equilibration of the occlusion and thorough subgingival curettage and portepolishing.

The wire splint was maintained in position on the teeth throughout the course of treatment. It was removed after four months.

Roentgenographic examination after four months and again after fifteen months showed an increase in the radiopacity of this region (see Figures 2 and 3). When the patient was last examined, the teeth were firm, the gingival tissue was a healthy pale pink, swelling was entirely absent and pockets were no longer present.

DISCUSSION

It is somewhat unusual for chronic radiolucent areas resulting from long standing trauma to show response so rapidly. In the author's

* Assistant Professor of Periodontia and Oral Medicine, New York University College of Dentistry.

Fig. 1

Fig. 2

Fig. 3

experience, roentgenographic changes of this type are rather more common when the injury results from a sudden blow, fall, etc.

Teeth which are in premature contact are subjected to excessive pressure during movements of the mandible. Pressure on teeth beyond physiologic limits results in resorption of alveolar bone.

As Miller[1] has shown, if the causative agents responsible for the alveoloclasia can be eliminated, the natural tendency is toward a return to a healthy condition. Having accomplished reattachment and the regeneration of the matrix, bone regeneration is possible.

This case also demonstrates the need for frequent roentgenographic examinations. It emphasizes the need for an understanding of the concept of occlusal equilibration and the institution of proper periodontal treatment as soon as degenerative changes are noted.

The reactivity of acutely inflamed tissue is probably better than in chronic cases. The actual periodontic treatment in most cases probably simulates to some extent the reactivity induced by an inflammatory reaction.

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Medical Views on Oral Problems

TRANSMISSION OF INFECTIOUS HEPATITIS*

To the Editor:—*A 4-year-old boy was bitten by a dog; the wound was cauterized, and a tetanus toxoid booster injection given. About six to seven weeks later the child became acutely ill, being given a diagnosis of infectious mononucleosis associated with hepatitis. After a stormy course of seven weeks the patient improved with supportive treatment. About 10 months later his 9-year-old brother was bitten by a dog from the same neighborhood. The wound was treated with an antiseptic, and a tetanus toxoid booster injection given. About six to seven weeks after this bite the brother became ill with hepatitis. The child also improved in seven weeks with supportive treatment. What is the relationship between the above illness and the dog bites, and what are the probable etiological factors and sequelae?*

M.D., Connecticut.

Answer.—It can be categorically stated that no cases of hepatitis have been reported from dog bites. "Infectious hepatitis" of dogs is not due to the same virus which produces infectious hepatitis in humans. Infectious mononucleosis has also never been reported to be produced by dogs. If there is any causal relationship between the hepatitis and the dog bite, then one would have to assume that the hepatitis followed the tetanus injection, due to the use of a contaminated needle or syringe. If the diagnoses of infectious mononucleosis and of infectious hepatitis were correct, then there can be no connection between these two cases. It is possible, however, that the second child actually had mononucleosis or that both children had infectious hepatitis. In the latter case, the second child might have contracted the virus from the first, provided that the first child continued to be a fecal carrier for a 9-or-10-month period. Such cases have been demonstrated, but probably occur only when persistent or chronic disease is present. The sequelae of such cases as are described are the same as with the ordinary case of infectious hepatitis. Recovery is generally complete in children of this age. However, it is probable that a certain small percentage develop chronic liver disease, and a few cirrhosis of the liver of a progressive nature.

* Queries and Minor Notes, J.A.M.A., Dec. 7, 1957, p. 1893.

COATING OF TONGUE*

To the Editor:—*A 55-year-old man states that for six months he has had a thick white coating on the posterior portion of his tongue. He scrapes off what he calls "granular material" with a spoon. Please make suggestions as to diagnosis and treatment.*

Alfred J. Berger, M.D., New Britain, Conn.

Answer.—The coating or furring on the tongue is not a sign of any specific disease. It is often associated with several conditions. Infections of the mouth and respiratory tract, even when not accompanied by fever, may be responsible for furring; mouth breathing, tobacco smoke, and febrile conditions are also associated with furring, but not gastrointestinal disease unless there is an accompanying fever.

Answer.—The epithelium of the tongue is constantly renewed and the desquamated outer layers are removed by its normal activity and environment. During the night when the activity of the tongue is greatly decreased, there is a tendency for a coating to develop. This coating usually disappears in the morning with resumption of greater activity on the part of the tongue, such as eating and talking. On this basis any decrease in oral activity due, for example, to a painful oral lesion may produce a coated tongue. The same may happen when there is a decrease in the moisture of the mouth caused by mouth breathing, diminished flow of saliva, or dehydration.

Another cause of coating of the tongue is alteration of the oral bacterial flora by antibiotics. When patients are given antibiotic drugs, particularly penicillin or chlortetracycline, the susceptible organisms in the oral flora are greatly diminished in number, permitting the resistant organisms to flourish and to alter the normal environment of the tongue. This is also known to occur lower in the gastrointestinal tract.

A third type of coated tongue is produced by functional or organic disturbances of the digestive tract, which lead to reversed peristalsis and the deposition of small particles of partly digested food from regurgitated gastric contents. Such regurgitation can take place at any time but is particularly likely to occur during the night when the individual is in the prone position. This type of coated tongue is sometimes called "worried" tongue. It can be detected by swallowing at night a gelatin capsule filled with lycopodium seeds and looking the next morning under the microscope for the characteristic seeds in scrapings from the tongue.

The most effective treatment of coated tongue consists in correction of the underlying local or systemic condition. If this is not known, the tongue can be scraped, e. g., with a toothbrush. In addition,

normal oral activity and hygiene should be promoted. In stubborn cases a 10% alcoholic solution of salicylic acid followed by 3% hydrogen peroxide may be tried.

* Queries and Minor Notes, J.A.M.A., Nov. 16, 1957, p. 1515.

TREATMENT OF CONGENITAL MACROGLOSSIA*

TO THE EDITOR:—*What is the best treatment for congenital macroglossia in a baby 4 months old? If surgery is indicated, when is the best time to operate?*

M.D., Mexico

ANSWER.—The macroglossia is either on a muscular basis or on a lymphangiomatous basis. The type of treatment will depend upon the size of the tongue itself and its resultant effect upon the nutrition of the child, i. e., whether or not the child is able to nurse or take its feeding, and also upon whether the condition is unilateral or bilateral. If the amount of the enlargement does not interfere with the child's feeding, then it is advisable to delay any active surgery for reducing the size of the tongue until a more opportune time. If, however, the nutrition of the child is impaired as a result of the increased size of the organ, treatment should be undertaken as soon as is feasible in order to permit the child to take its nourishment.

If the lesion is on a vascular basis, some have advised the use of radium. Radium is effective in a capillary type of lesion but seldom in a cavernous type of lesion. Some have advised and used repeated fulgurations or electrocoagulation in order to minimize the amount of surgical shock. This method has been used very effectively. It is not effective in the muscular type of macroglossia, nor is the use of radium effective in the muscular type. Marginal excision of the tongue in both the lymphangiomatous and muscular types has given excellent results. If the lesion is unilateral, then it may be advisable to ligate the lingual artery before attempting the excision. In this manner the amount of bleeding is reduced to a minimum. In a bilateral case, ligation of the lingual arteries is contraindicated.

* Queries and Minor Notes, J.A.M.A., Dec. 28, 1957, p. 2243.

RECURRENT PAROTITIS*

TO THE EDITOR:—*A woman aged 55 years, during the past six years has had frequent attacks each year of parotitis. Usually only one of the parotid glands is involved, but a few times both. The glands become swollen, painful, and tender, and the patient has moderate fever. Radiograms do not show calculi in the parotid glands or Stensen ducts. There is no cervical adenopathy. Several years ago the patient had a thyroidectomy because of a recurrent simple thyroid condition. With each attack the patient is given penicillin, and recovery is made after five or six days. What is the cause and what treatment would be effective in prevention of the frequent recurrences of parotitis?*

A.M. Loes, M.D., Dubuque, Iowa.

ANSWER.—Sialography can be helpful in establishing the diagnosis in this case, especially to rule out inflammatory stenosis. Sometimes the type of parotid swelling described is caused by a tiny stone near the orifice of the parotid duct, even though x-ray films do not show calculi. If such a stone is present in this location, it can sometimes be seen by dilating the duct orifice with a punctum dilator, provided that loupe magnifying glasses and a headlight are used for the inspection. In case no stone is present, the next most likely diagnosis is a mass of thick mucus in the duct system causing obstruction.

In either case, dilating the orifice of the parotid duct with a punctum dilator and the passage of lacrimal probes periodically is often curative. But if any stone is present, it must be removed sooner or later. The orifice of the duct can be split, with the patient under local anesthesia, for 2 or 3 mm., or enough to allow extrusion of such a stone. This can be done as an office procedure.

This consultant has had several unilateral cases (with negative x-rays for calculi) in which a stone of perhaps 1 mm. or so in diameter was found; this had acted as an obstructive ball-valve. In each case cure resulted from the measures outlined. Good mouth hygiene is important in prophylaxis and can be aided by alternating mouthwashes of aqueous 2% boric acid, 10% glucose, and dilute milk of magnesia.

* Queries and Minor Notes, J.A.M.A., Dec. 21, 1957, p. 2139.

LOSS OF TASTE AFTER TONSILLECTOMY*

TO THE EDITOR:—*Loss of taste has occured in a patient after tonsillectomy. She is a cook, and taste is very essential in her business. Please make suggestions as to further treatment. How long will this condition persist?*

M.D., Washington.

ANSWER.—Loss of sense of taste as a complication of tonsillectomy is indeed a rare condition. Probably some injury was done to the glossopharyngeal nerve, which supplies taste fibers to the posterior portion of the tongue. However, the anterior two-thirds of the tongue is supplied by the chorda tympani, and certainly could not have been injured during the tonsillectomy. The glossopharyngeal nerve also supplies sensation to the mucous membrane of the pharynx, the fauces, and the palatine tonsil. It is conceivable that some scar tissue may

have grown over from the tonsil fossae and areas immediately adjacent to the dissected areas of the tonsil, especially at the lower poles of both tonsils.

A careful inspection of the areas which were operated on is recommended, especially near the posterior portion of the tongue; with the patient under a little local anesthesia, perhaps some of the scar tissue could be removed. Also, the use of vitamin B_{12} injections several times a week and the local application of 95% alcohol to stimulate the taste buds might be considered. In general, the sense of taste should return within 18 months of the time of operation, if there is no central lesion of the brain causing loss of sense of taste and other complications.

* Queries and Minor Notes, J.A.M.A., Dec. 7, 1957, p. 1898.

CARCINOMA OF PAROTID GLAND*

TO THE EDITOR:—*A patient had a small, pea-sized tumor removed from the lower pole of the parotid gland, which was reported to be an adenocarcinoma. After six months there is an apparent recurrence of the tumor at the site of the previous enucleation. The tumor mass, on palpation, is about 1 cm. in diameter. Should the entire parotid gland be removed, sparing whatever branches of the seventh cranial nerve that are not involved in the tumor? Should a radical parotidectomy be done, removing the entire gland and all the branches of the seventh nerve? Should only the lower pole, which contains the tumor only, be removed, sparing all the seventh nerve branches possible?*

M.D., New York

ANSWER.—The entire parotid gland should be removed without consideration of the facial nerve in any way whatsover. The malignant tumor has recurred, and it is particularly necessary to remove the parotid lymph nodes lying at about the junction of the superficial and deep lobes of the parotid gland, together with all other parotid nodes. There is a remote possibility of identifying the trunk of the facial nerve after its emergence from the stylomastoid foramen, where it lies free for about 6 to 12 mm., so that the nerve can be followed forward to where it encircles the isthmus of the superficial and deep lobes of the parotid. If so, the upper division of the pes anserinus can be spared, thus enabling the patient to close his eye. This point is of minor importance because the tumor may be already lethal. Every effort should be made to excise it, with at least a centimeter of normal tissue around it, whether or not this damages any or all of the facial nerve.

Furthermore, a careful check should be made for metastatic disease of the lymph nodes in the neck and for more distant metastases. If a suggestive node is found in the neck, a block dissection should be done along with the radical parotidectomy.

* Queries and Minor Notes, J.A.M.A., Dec. 14, 1957, pp. 2033-34.

RADIUM TREATMENT OF CANCER OF THE TONGUE*

The author reviews the therapy of cancer of the tongue on the basis of observations of 100 patients with lingual cancer observed by him between 1933 and 1951. Eighty were men and 20 were women; 78 were between 46 and 66 years of age. Sixteen patients were not treated or did not complete the course of therapy. Thirty-six had been treated previously: 17 with caustics, 8 with radium or roentgen therapy, 7 with surgical treatment, and 4 with antisyphilitic treatment. Radium therapy is now considered the best method of treating the primary lesion. The author's technique consists of 3 parts: (1) the surface application of about 500 mc. of radon in a silver applicator (or cobalt-60 in equal strength) over different sections of the periphery of the tumor, with 1 or 2 areas treated for 5 minutes daily until about 6,000 r of gamma rays have been delivered to the surface of the growth; (2) the implanation of gold (or lead) radon seeds, each having a strength of 0.5 mc., throughout the base of the tumor; and (3) the bombardment of the lymph-node-bearing area, or of palpable lymph nodes if present, at a distance of 2 to 6 cm. with a radon applicator containing from 500 to 1,000 mc. daily until at least 5,000 gamma r have been given. If palpable nodes persist, a block dissection on the side of the nodes is performed.

Nineteen of the cases were considered indeterminate, and of the 81 determinate cases 31 were considered "favorable" and 50 "unfavorable." Of the favorable group 20 patients were alive and free of disease after 5 years. Of the unfavorable group, 5 were cured. The percentage of cures in determinate cases was 30.9%. The importance of early diagnosis is borne out by the fact that in 64.5% of favorable cases a complete recovery was obtained, while only 10% of patients with advanced lesions were cured. Any firm swelling of the tongue should be considered malignant unless proved otherwise. The same rule applies to chronic ulcers. As the great majority of tumors developed on the lateral border of the tongue or at the top, and as many patients had jagged or sharp teeth, the author believes that chronic irritation from the teeth is one of the most important etiological factors. Good dental care with the rounding off of sharp edges of worn teeth is basic prophylaxis. As some patients were heavy smokers or chewed tobacco and the growth developed either where the stream of smoke hit the tongue or adjacent to the cud, it is thought that tobacco played an etiological role.

* By J. E. Breed, Radiology 69:214-223 (Aug.) 1957, (Syracuse, N.Y.) Medical Literature Abstracts, J.A.M.A., Dec. 14, 1957, pp. 2025-26.

Book Reviews

Einfuhrung in die Zellular-therapie (Introduction to Cellular Therapy), by Dr. Paul Niehans, Verlag Hans Huber, Bern and Stuttgart, 1957. Price SFR. 12.50.

This small volume contains a series of lectures by Dr. Niehans regarding his experiences with cellular therapy. The technique, introduced by the author, consists briefly, of the use of fetal animal cells injections which may, according to the author, replace or substitute for diseased organs of the patient. "Cellular therapy is directed towards the development of poorly formed or the regeneration of diseased organs"; thus in the treatment of myocardial damage, fetal animal heart muscle cells and placental cells are injected; in nephrosis, fetal kidney cells and placental cells are injected.

Dr. Niehans points out that in this fashion, injection of fetal cells acts similarly to a transplantation of organs, thus reducing the necessity of continuous substitution therapy one must maintain when organ extracts or hormones are used. Included in these lectures are a series of case reports of patients treated by this method with apparent success.

The discussion of cellular therapy was of interest to this reviewer since to my knowledge little is known in this country regarding this procedure. Although some American practitioners are utilizing this form of therapy, controlled reports are still not available.

Of particular challenge to dentists is Dr. Niehans' comment regarding the possibility of using cellular-therapy in dentistry which the author feels should be attempted.

Dr. Niehans concludes his remarks with "Gentlemen, here are many opportunities for future investigations. Every advance is a realized utopia. May you be successful in realizing many utopias." This wish should be furthered by scientific evaluations of cellular-therapy.

S. S. STAHL

Implant Dentures, by Aaron Gershkoff, B.S., D.D.S. and Norman I. Goldberg, D.D.S., 256 pages, 327 illustrations, J. B. Lippincott Co., Philadelphia, 1957, $12.00.

This book is devoted entirely to Implant Dentures, and it is probably the first full text on the subject. Since it is a first, comparison with other books of a similar type is not possible.

Chapters on the 'History of Metal Appliances in the Body' and 'The History and Development of Implant Dentures' is therefore of great significance.

Important portions of the book deal with the edentulous mouth, its anatomical relations, its problems as they relate to the indications and contra-indications of Implant Dentures.

The bulk of the book deals with the techniques and procedures of making implant dentures and the material presented is quite complete. It is to the credit of the authors that they present case failures together with the reasons for the difficulties encountered.

Histopathology of the involved areas is presented to augment the case for this type of prosthetic appliance. This is done for both the hard and soft tissues of the jaws.

A number of case histories are included dating from 1948. These are of interest from the point of view of the application of the principle of this type of denture in regard to denture retention and tissue tolerance and reaction.

The book is well written. Bold type on coated stock makes for easy reading. The illustrations are well reproduced and the other mechanics of the book are good. For those interested in Implant Dentures, this is a worthwhile text.

LOUIS R. BURMAN

Accepted Dental Remedies, Published by the American Dental Association, 23rd Edition, Chicago, 228 pages, 1958, $3.00.

Accepted Dental Remedies 1958 represents a new look at the drugs used in the present day practice of dentistry and the problems which face the dentist. The material in this edition not only has been brought up to date but it has been reorganized in order to make the book easier for the busy practitioner to use. A greatly expanded table of contents provides an outline of the important sections of the entire book.

This small, soft covered book contains 228 pages, including the provisions for acceptance of products, a general index, a distributor's index, an index of current reports from the Council and an index of more recent reports on products not listed in Accepted Dental Remedies, including those classified in Groups B, C and D.

This excellent handbook is now available from the Order Department of the American Dental Association.

Full Dentures, by Chester Landy, D.M.D. C. V. Mosby Co., St. Louis, 182 pages with 82 illustrations, 1958, $5.85.

Dr. Landy has taken into consideration that time does not permit the busy practitioner to evaluate and clinically apply all the techniques which have been advocated in full denture construction. There is a definite need today to bridge the gap between the exhaustive text and the short scientific article. Dr. Landy has written a text which is small in size but readily available as an excellent reference and guide.

The reader may accept Dr. Landy's method of full denture instruction in its entirety, or may adapt certain phases to improve his own technique. The clinical use of non-anatomic teeth and the mucostatic or minimal pressure impression technique are thoroughly discussed. Denture base materials, the adjunct use of drugs, the single denture, the all important post insertion adjustments, and the practical psychological approach to the patient's problems, are competently handled by the author.

Selected readings or references are mentioned for each chapter in addition to the general bibliography. This compact text is a welcome addition to prosthetic dentistry literature.

Clinical Applications of Hypnosis in Dentistry, by S. Irwin Shaw, D.M.D., M. Ed. W. B. Saunders Co., Philadelphia, 173 pages, 1958, $4.50.

During the last decade there has been a modern revival of interest in hypnotism and it has manifested itself most strongly in the dental field. There has been a need for a short, concise presentation of the fundamental techniques of hypnotism within the framework of applications. This text is not only for the dental practitioner but also for any other professional person actively engaged in the use of hypnotism.

This treatise has many worthwhile features in addition to its descriptions of techniques. The author is fully aware of the dangers of misapplication and has carefully limited the field of application of hypnosis for the dentist. The author shows the reader in simple language why psychological factors in everyday living and dental practice cannot be ignored in the effective use of suggestions and how these factors can be used to best advantage. Dr. Shaw goes on to show the reader how he can use the principles of suggestion as a psychological tool in his relationships with his patients.

Those who are interested in hypnosis, no matter what their field of specialization may be, will gain much from reading this new text.

EDUCATION COMMITTEE

The Education Committee of the American Academy of Dental Medicine will sponsor a registered clinic at the Twelfth Annual Meeting in Montreal on Friday, May 30, 1958 directly after the Award Luncheon. Dr. Philip Ament will present a clinic entitled, "Hypnoanesthesia for the Dentist and Physician".

In conjunction with this presentation, Dr. Ament will show two motion pictures, one for the dentists and one for the physicians. The Education Committee believes that this presentation will be of timely interest to all our members.

The fee for this registered clinic will be five dollars. Please send your reservation and check to Dr. Harold E. Brennan, Chairman, 400 Madison Avenue, New York 7, N.Y.

Academy Proceedings

AMERICAN ACADEMY OF DENTAL MEDICINE ANNOUNCES MID-ANNUAL MEETING

The American Academy of Dental Medicine will hold its Thirteenth Mid-Annual Meeting and Luncheon at the Hotel New Yorker, New York City, on Sunday, December 7th, 1958. There will be a business meeting for members at 10:00 A.M. This will be followed by a luncheon and scientific session for Academy members and friends starting at 12:30 P.M.

The afternoon scientific session will be devoted to "The Gingival Tissue." Participating in this program will be:

1. Henry M. Goldman, D.M.D., Director of the Riesman Dental Clinic, Beth Israel Hospital, Boston; Professor of Periodontology and Chairman of the Department of Periodontology, Graduate School of Medicine, University of Pennsylvania.
2. Maury Massler, D.D.S., Professor and Chairman of the Department of Pedodontics, University of Illinois.
3. Stanley D. Tylman, D.D.S., Professor and Chairman of the Crown and Bridge Department, University of Illinois.

All members and interested dentists and physicians are cordially invited to attend. For reservations and programs address the Chairman, Dr. Nathan Wachtel, 36 Central Park South, New York, N. Y. or Academy Secretary, Dr. George J. Witkin, 501 Madison Avenue, New York, N. Y.

NEW JERSEY SECTION

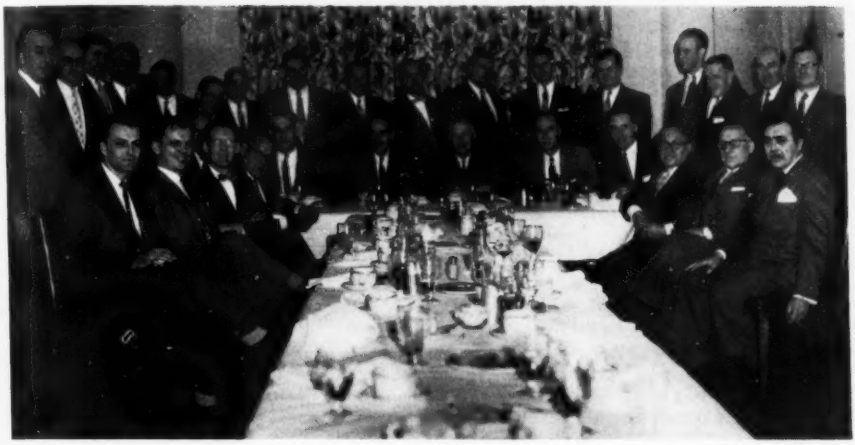
The New Jersey Section of the American Academy of Dental Medicine will hold its next meeting on Wednesday, April 23, 1958 at 1:00 P.M. at the facilities of the Martland Medical Center, 116 Fairmont Avenue, Newark, New Jersey. The clinician for this meeting will be Dr. Martin S. Protzel, a highly qualified and competent teacher. Dr. Protzel is Assistant Professor of Oral Pathology at New York University College of Dentistry and a Diplomate of the American Board of Oral Surgery. His topic will be "Clinical and Laboratory Diagnostic Procedures."

The program will consist of the following phases:

1. General business meeting at 1:00 P.M.; introduction of Clinic Program in the Amphitheatre at 1:30 P.M.
2. Patients will be presented with oral lesions of local or systemic character.
3. Symptoms and signs associated with the lesions will be discussed and all the pertinent laboratory procedures will be evaluated.
4. A biopsy of a lesion will be taken when indicated and by means of quick frozen section technique, an immediate histo-pathological diagnosis will be made.
5. Microscopes shall be available for those in attendance to review slides.
6. Summary and discussion of oral lesions examined with additional information on laboratory procedures.

REUBEN FELTMAN, D.D.S., *Program Chairman*

MONTREAL SECTION



Members of the Montreal section of the American Academy of Dental Medicine pose for the above photograph taken on January 15, 1958 at the Reform Club in Montreal where they met to hear an address by Dr. Z. Metzl, M.D., D.D.S., Associate Professor at the University of Montreal.

Seated at the Head Table, (L. to R.) H. T. Oliver, Secretary of the Montreal Section; Dean J. McCutcheon of McGill University Dental Faculty; Dr. Marcel B. Archambault, President, the American Academy of Dental Medicine; Dr. Z. Metzl; Dr. A. Scherzer, President of the Montreal Section and Dr. Guy Langellier, Vice President, Montreal Section.

NEW YORK SECTION

A meeting of the New York Section of the American Academy of Dental Medicine was held on February 27, 1958 at the George Washington Hotel, New York City.

The speaker of the evening was Dr. John D. Piro who spoke on the "Effects of Irradiation on Bone and Teeth and Cases of Special Interest in Relation to Metastises of Cancer to the Jaw." Dr. Piro is Attending Dentist at Francis Delafield Hospital and Instructor in Dentistry at Columbia University. The talk was most stimulating and provoked active discussion.

Dr. Sidney Sorrin, Associate Professor and Acting Chairman of the Department of Periodontia and Oral Medicine, New York University College of Dentistry, addressed the membership prior to the scientific meeting. He delivered a most heartfelt eulogy for his beloved friend and colleague, Dr. Samuel Charles Miller. He read a very beautiful, original poem, dedicated to Dr. Miller's memory, after which he called for a minute of silence. It was a very moving tribute to a truly great man.

Our Section was honored by the presence of our National President, Dr. Marcel Archambault of Montreal, who brought greetings and words of encouragement from the National Organization.

An executive committee meeting was held at the conclusion of the general meeting. Plans for the coming year were initiated, with the formation of committees and the appointment of Chairmen. A slate of officers was nominated which will be presented to the membership at the next meeting.

Howard L. Ward, *Section Editor*

Announcements

POSTGRADUATE TRAINING IN PERIODONTIA AND ORAL MEDICINE NEW YORK UNIVERSITY COLLEGE OF DENTISTRY

The following postgraduate courses in Periodontia and Oral Medicine will be offered at the New York University College of Dentistry and will be given by Dr. Sidney Sorrin and associates:

No. 124. REFRESHER COURSE IN PERIODONTIA AND ORAL MEDICINE
Ten sessions, Wednesdays, 4:00 to 6:00 P.M.
February 11, 1959 to April 22, 1959.
Tuition and books. $156.00. Instruments approximately $55.00.

Theoretical and practical application of periodontal diagnosis and techniques to general dentistry. Four lecture and six clinical demonstration sessions. Equilibration of occlusion is performed by the student on casts. Patient demonstrations include equilibration of occlusion, conservative and radical therapy, home care, etc.

No. 123. PERIODONTIA AND ORAL MEDICINE IN THE SPANISH LANGUAGE
Four weeks, Monday through Friday, 9:00 A.M. to 4:00 P.M.
March 2 to March 27, 1959 inclusive
Tuition including books and instruments approximately $385.00.

Theoretical and practical application of Periodontia and Oral Medicine is fully explained and demonstrated. Equilibration of occlusion is performed by the student on casts. Lectures and clinical demonstrations of diagnosis and treatment planning, conservative and radical techniques, equilibration of occlusion, home care, etc. The entire course is conducted in the Spanish language.

No. 122. COMPREHENSIVE PREPARATION IN PERIODONTIA AND ORAL MEDICINE

No. 122 A. One academic year, full time. September 17, 1958 to May 22, 1959.
Tuition $1,010.00. Instruments and books approximately $135.00.

No. 122 B. Two academic years, full time. September 17, 1958 to May 27, 1960.
Tuition $2,020.00 (two years). Instruments and books approximately $231.00.

No. 122 C. Two academic years, half time. September 17, 1958 to May 27, 1960. (Two and one half days weekly.)
Tuition $1,010.00 (two years). Instruments and books approximately $135.00.

No. 122 D. Four academic years, half time. September 17, 1958, to May 25, 1962. (Two and one half days weekly.)
Tuition $2,020.00 (four years). Instruments and books approximately $231.00.

These courses are desgined for comprehsensive preparation in Periodontia and Oral Medicine and are of particular significance for certification towards specialization. All didactic and clinical phases are covered with the objective of developing expert proficiency.

No. 222. GRADUATE COURSE IN PERIODONTIA AND ORAL MEDICINE.
Two academic years, full or half time, beginning Sept. 17, 1958. Tuition $2,020.00. (two years). Instruments and books approximately $275.00.

This program leads to the degree of Master of Science (in Dentistry). The objectives are to teach superior proficiency in Periodontia and Oral Medicine based on adequate background in the basic sciences.

DOCTORS' ORCHESTRAS

Dentists and members of allied professions are invited to become members of two doctors' orchestras in New York City. Non-medical musicians, may also join. The Doctors' Orchestral Society of New York rehearses Thursday evenings in the auditorium of the Stuyvesant High School, 15th Street and First Avenue, New York City at 8:30 P.M. The Brooklyn Doctors' Symphony meets Wednesday evenings in the auditorium of the Brooklyn High School of Homemaking at 901 Classon Avenue, corner of Washington Avenue, Brooklyn at the same time. Both groups are rehearsing now.

For further details contact: Dr. Benjamin A. Rosenberg, 909 President Street, Brooklyn, New York, NE 8-2370.

TUFTS UNIVERSITY SCHOOL OF DENTAL MEDICINE

The following courses listed below will be presented at Tufts University School of Dental Medicine during 1958. Application and further information may be obtained by writing the Director, Division of Graduate and Postgraduate Studies. Tufts University School of Dental Medicine, 136 Harrison Avenue, Boston 11, Mass.

DPG. 802—ORAL ROENTGENOLOGY
Tuesday and Wednesday
April 22, 23, 1958
Tuition $50.00 — Class limited
Dr. John H. Barr, Professor of Oral Diagnosis and Roentgenology

DPG. 902—HYPNOSIS AND ITS CLINICAL APPLICATIONS
A Course for Dentists, Physicians and Psychologists
Friday, Saturday, Monday, Tuesday and Wednesday
May 9, 10, 12, 13, 14, 1958 — 9 a.m. to 5 p.m.
7 p.m. to 9 p.m.
Tuition $150.00 — Enrollment Limited
Dr. Lawrence M. Staples, D.M.D.
Boston, Massachusetts
Dr. Milton H. Erickson, M.D.
Phoenix, Arizona
Dr. Jacob Seidenberg, D.M.D.
Chelsea, Massachusetts
And Staff

DPG. 704—FULL DENTURE PROSTHESIS
One Week — June 2 through June 6, 1958
Tuition $150.00 — Class limited to seven
Dr. Irving Hardy, Professor of Graduate and Postgraduate Prosthetics

DPG. 501
CLINICAL DENTISTRY FOR CHILDREN
Monday, Tuesday and Wednesday
June 9, 10, 11, 1958
Tuition $75.00 — Class limited to 12
Dr. Frederic Shiere, Dr. Helmi Fogels, Dr. Harold Berk, and Dr. Philip McCaul

DR. IRVING R. HARDY TO BE HONORED

The Faculty of Tufts University School of Dental Medicine announces a testimonial dinner for Dr. Irving R. Hardy, Professor of Graduate and Postgraduate Prosthetics, to be held Wednesday Evening, April 30, 1958, on the campus of Tufts University, Medford, Massachusetts.

Dinner :—7:00 p.m., Jackson Gymnasium
Cocktails :—6:00 p.m., Alumnae Lounge
Subscription:—$10.00—Individual
—$20.00—Couple

Reservations are limited and must be made on or before April 10, 1958. Dress Informal. Please make checks payable to: The Dr Hardy Testimonial Dinner.
Mail reservations to: Dr. L. Walter Brown, Jr., Chairman, Prosthetic Dentistry Department, 136 Harrison Avenue, Boston 11, Massachusetts.

AMERICAN DENTAL SOCIETY OF ANESTHESIOLOGY

The New York State Dental Society of Anesthesiology, a component of the American Dental Society of Anesthesiology, is having its Spring Meeting Sunday Afternoon May 11, 1958 at 2:00 P.M. in the Boston Room of the Hotel Statler, Buffalo, N. Y. The meeting will consist of a business and scientific session. The topic for the scientific session will be "Physiological Derangements in Anesthesia." The essayist will be Samuel L. Lieberman, M.D., F.A.S.A., Diplomate, American Board of Anesthesiologists. All members of the Dental and Medical Societies are invited to attend.

UNIVERSITY OF ALABAMA SCHOOL OF DENTISTRY

During the month of May, the University of Alabama School of Dentistry will present three refresher courses; Victor H. Sears, D.D.S., will present a course in *Complete Denture Prosthesis* on May 1, 2, and 3, 1958; Harry Sicher, M.D., D.Sc., will conduct a program in two parts, *Surgical Anatomy of the Head and Neck* (Part I) on May 17, 18, and 19, 1958 to be followed by *Etiology and Diagnosis of Pain of Dental Origin* (Part II) on May 20 and 21, 1958; and Samuel D. Gore, D.D.S., will give a course in *Removable Orthodontic Appliance Construction* on May 24, 25, and 26, 1958. The latter course is limited to those practicing orthodontics as a specialty.

For further information please write to the Director, Refresher Course Program, University of Alabama School of Dentistry, 1919 Seventh Avenue South, Birmingham, Alabama.

TEMPLE DENTAL ALUMNI TO MEET

The 94th Annual Reunion of the Temple University Dental School Alumni will take place at the Drake Hotel in Philadelphia on April 30, 1958. Members of the classes of 1908, 1913, 1918, 1923, 1928, 1933, 1938, 1943, 1948 and 1953 will meet in reunion and will be contacted by their respective committees.

NINTH ANNUAL BERKSHIRE CONFERENCE IN ORAL PATHOLOGY AND PERIODONTOLOGY

The Division of Graduate and Postgraduate Studies of Tufts University School of Dental Medicine will conduct the Ninth Annual Berkshire Conference in Oral Pathology and Periodontology at Lenox, Mass., June 15-19, 1958. The Conference will consist of four days of lectures, seminars and panel discussion regarding the clinical problems by outstanding teachers and clinicians in dentistry, medicine and their related fields.

The curriculum is arranged so that the mornings and evenings are devoted to professional activities with afternoons open for recreation. This is a conference for General Practitioners as well as those interested in a specialized practice.

The Central Theme of the Conference will be "Gerodontics—Oral Problems Associated with Aging."

Included amongst the faculty and subjects to be considered are:

DR. JOSEPH L. BERNIER—Armed Forces Institute of Pathology, Washington, D.C.
Subject: CLINICO-PATHOLOGICAL SEMINAR ON ORAL MUCOUS MEMBRANE PROBLEMS OF THE AGED

DR. IRVING GLICKMAN—Tufts University School of Dental Medicine, Boston, Mass.
Subject: FUNCTIONAL CONSIDERATIONS WHICH GOVERN BONE LOSS IN PERIODONTAL DISEASE

DR. MAX GRATZINGER—Chicago, Illinois
Subject: OCCLUSAL AND FUNCTIONAL CONSIDERATIONS IN CLINICAL PROCEDURES

DR. BERNARD JANKELSON—University of Washington School of Dentistry, Seattle, Washington
Subject: THE NATURE OF THE DENTITION—ITS ROLE IN THE STOMATOGNATHIC SYSTEM AND THE EFFECTS OF AGING ON THE MORPHOLOGY OF THE TEETH-ABERRATIONS OF FORM AND THEIR SIGNIFICANCE AND CLINICAL PROCEDURES IN THE ADJUSTMENT OF OCCLUSION

DR. HERBERT MESCON—Boston University School of Medicine, Boston, Mass.
Subject: BASIC ASPECTS OF ALLERGY AND THEIR SIGNIFICANCE IN ORAL MUCOUS MEMBRANE DISEASE

DR. EDWARD J. STEIGLITZ—Washington, D.C.
Subject: MEDICAL PROBLEMS OF THE AGED AND THEIR SIGNIFICANCE IN DENTISTRY

DR. JOSEPH P. WEINMANN—University of Illinois Dental School, Chicago, Illinois
Subject: CHANGES IN THE PERIODONTAL TISSUES WITH AGING AND THEIR CLINICAL SIGNIFICANCE

For further information and application, write to: Ninth Annual Berkshire Conference, Tufts University School of Dental Medicine, 136 Harrison Avenue, Boston 11, Massachusetts.